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LEWIS A. CONNER . . . . . Editor

Associate Editors

HUGH MCCULLOCH EVELYN HOLT

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Original Communications

A FURTHER REPORT ON THE PROGNOSIS OF ANGINA PECTORIS AND OF CORONARY THROMBOSIS. A STUDY OF FIVE HUNDRED CASES OF THE FORMER CONDITION AND OF TWO HUNDRED CASES OF THE LATTER*

PAUL D. WHITE, M.D., AND EDWARD F. BLAND, M.D.
BOSTON, MASS.

SINCE the time of Heberden's classical description of angina pectoris in the eighteenth century, and doubtless for centuries before, the uncertainty of the prognosis has remained one of the outstanding features of this condition. The frequent association of angina pectoris with sudden death has made the two almost synonymous in the minds of many patients who feel that an early end is inevitable; too often such a gloomy outlook has been shared also by the physician in charge, who, perhaps with a fatalistic point of view, makes no attempt to determine the patient's chances for survival for any length of time, as for instance five years or more. But even though it is recognized that patients may live for many years after the onset of symptoms, an intelligent prognosis in the individual case still remains one of the most difficult problems in medicine. We have been very much interested in trying to find out if there are any helpful clues in this difficult task; as a result of our study we are confident that there are such clues even though there is a great deal still to learn. The tremendous importance of the subject is indicated by two facts; first, angina pectoris is one of the most common serious medical complaints today and apparently is becoming steadily more common; and second, it is particularly likely to attack people in important professional, administrative, and business positions who need to know something at least of their chances for life and work in the future.

In the present investigation we have studied a group of 500 consecutive cases of angina pectoris and 200 consecutive cases of coronary thrombosis seen in private practice during the past ten years in order to determine what the expectation of life in the two conditions

*Read before the American Heart Association at Philadelphia, Pa., June 4, 1931.

may be, and favorable and unfavorable factors the presence of which may be of good or of ill omen for the patient. A preliminary report of the first 200 cases of angina pectoris and of 62 cases of coronary thrombosis was published five years ago.[1] The present paper includes a follow-up report of these cases and is in the nature of an intermediate study of the problem. After another five years or more we shall publish a more or less final report of these groups of patients.

PROGNOSIS IN THE PRESENT SERIES OF FIVE HUNDRED CASES OF ANGINA PECTORIS

All patients included in this series had paroxysmal oppression in the front of the chest, generally substernal, often radiating to the left arm or both arms, rarely to the right arm alone, brought on particularly by exercise, especially after eating, in cold weather, or when hurried, and relieved within a few minutes by rest or by nitrites. Many of the patients were nervous, but no attempt was made to separate "primary" from "secondary" angina pectoris; such a separation we believe is unwise, but as in the previous study, other varieties of chest or heart pain were carefully excluded from the present series of real angina pectoris.

TABLE I

PROGNOSIS OF ANGINA PECTORIS AND OF CORONARY THROMBOSIS

1. *Angina pectoris*—500 cases (including follow-up of 200 cases previously reported)	
213 cases known to be dead—average duration of life after the onset of angina pectoris	= 4.4 years
273 cases known to be living—average duration of the angina pectoris	= 5.1 years
486 cases—average	= 4.9 years
14 cases not traced	
Three patients had angina pectoris for over 20 years—two dead, one still alive over 21 years after onset of this trouble.	
2. *Coronary thrombosis* (acute)—200 cases (including follow-up of 62 cases previously reported)	
101 cases known to be dead—average duration of life after the acute cardiac infarction (range = a few hours to 11 years)	= 1.5 years
94 cases known to be alive—average duration since acute cardiac infarction (maximum = 17 years)	= 3.2 years
195 cases—average	= 2.4 years
5 cases not traced.	

Of the five hundred patients with angina pectoris upon whom this study has been made, recent reports were obtained upon all except fourteen whose present status remains undetermined. Two hundred and thirteen (213) patients are dead, while two hundred and seventy-three (273) are known to be alive. Of the 213 patients known to be dead, 9 lived less than two months. In the remaining group of 204

patients the average duration from the onset of the angina pectoris to death was 4.6 years. The average duration of life in the total group of 213 cases known to be dead was 4.4 years. The average duration of the angina pectoris to date in the 273 living patients is 5.1 years, which gives an average duration of 4.9 years for the entire series of living and dead patients. In our series three patients have had angina pectoris for twenty or more years; two of these patients are dead, both having had attacks of angina pectoris for twenty years; the remaining patient is living and fairly well in spite of the fact that he has had angina pectoris for twenty-one years. Mackenzie found the average duration of life after the onset of angina pectoris to be 5.4 years in 213 patients whom he was able to follow from the onset of symptoms to death.[2] It seems probable that upon completion of our series the average duration of life will exceed that reported by Mackenzie and it may surpass six years.

Although the conclusions arrived at from an analysis of a large series of cases of angina pectoris cannot be applied *in toto* to the individual case, it is instructive to determine the occurrence of various factors which may influence favorably or unfavorably the course of the disease. In Table II we have presented data concerning the sex, age at onset, history or presence of hypertension or syphilis, evidence of arteriosclerosis, occurrence of coronary thrombosis clinically diagnosable, enlargement of the heart, character of the heart sounds, congestive failure, radiation of the pain to the left, right, or both arms, the severity of the pain, presence or absence of heart-block, either auriculoventricular or intraventricular, the T-wave of the electrocardiogram (in Leads I and II), the degree of nervous sensibility, and the number of cases that showed a normal heart upon physical and electrocardiographic examination. In order to give the relative frequency of the factors mentioned above, we have arranged the cases in three groupings; (1) the total series, (2) 100 patients who died within three years after the onset of angina pectoris, and (3) 100 patients who lived more than seven years after the onset.

It has been possible to determine with a fair degree of accuracy the manner or cause of death in a considerable number of the series. Of the 213 patients who died, 62 died suddenly, certainly in a large majority of cases from angina pectoris itself; in 38 other cases death was attributed definitely to coronary thrombosis; 45 more cases succumbed to congestive failure; while in 31 others death was said to be of cardiac origin but the details were insufficient to make a definite diagnosis possible. Thus, it seems evident that in the majority of the group death could be attributed directly to failure of the heart, that is, in 176 patients (82 per cent) of the 213 of the completed series. Of the remaining patients who died, various conditions were responsible, including carcinoma, pneumonia, hemiplegia,

TABLE II

DATA OF FIVE HUNDRED CASES OF ANGINA PECTORIS

	TOTAL 500 PATIENTS	DEAD WITHIN 3 YEARS (100 PATIENTS)	LIVED OVER 7 YEARS (100 PATIENTS)
Sex:			
Male	376 (75 per cent)	81	77
Female	124 (25 per cent)	19	23
Age at Onset:			
Average	56.5	59	53
Limits	20-80	24-80	20-61
Hypertension (160/110)	182	42	34
Syphilis:			
Positive	22	6	3
Questionable	8	1	3
Negative	470	93	94
Arteriosclerosis:			
Positive	268	53	45
Questionable	50	18	18
Negative	182	29	37
Coronary thrombosis:			
Positive	130	34	19
Questionable	75	20	19
Negative	295	46	62
Cardiac enlargement:			
Positive	316	80	55
Questionable	21	3	3
Negative	163	17	42
Character of heart sounds:			
Good	274	44	64
Fair	154	34	12
Poor	72	22	24
Congestive failure:			
Positive	77	22	13
Negative	423	78	87
Pain referred to			
Left arm alone	177	30	45
Right arm alone	20	6	1
Both arms	172	38	29
Severity of pain:			
Uncertain	13	4	1
Mild	129	24	26
Moderate	151	31	39
Marked	104	18	18
Severe (decubitus)	103	23	16
Electrocardiogram:			
Heart-block: auriculo-ventricular			
Positive	11	2	2
Negative	382	69	33
Heart-block: intraventricular			
Positive	51	13	20
Negative	342	58	65
Inverted T deflection (Lead I or II)	58	17	10
Nervous sensibility marked			
Positive	239	40	52
Negative	227	46	31
Normal cardiac examination, blood pressure, and electrocardiogram	74	9	28

and postoperative complications. The average age at death for the group was 62.2 years, being slightly lower for the males (61.6 years) than for the females (64.7 years). Greater opportunity for rest in the case of women may account for this difference.

It has long been recognized that even in the presence of severe angina pectoris no abnormalities may be detectable by any method of examination. Of the group of 393 who had had electrocardiograms, 74 patients (19 per cent) showed normal hearts on physical examination (in size, sounds, rate and rhythm), normal blood pressure, and normal electrocardiograms. From Table II it will be seen that only 9 of these patients, or 12 per cent, had died in less than three years; while 28, or 37 per cent, had lived seven or more years since the onset of their symptoms, so that normal findings by the above methods of examination appear to be definitely favorable.

Fifty-eight patients of the 393 who were electrocardiographed showed an inversion of the T-wave in Leads I or II of the "coronary type," and from Table II it is seen that this occurred in 17 patients who lived less than three years after the onset of symptoms of angina pectoris, and in 10 who lived more than seven years; so that, although considered as evidence of coronary disease and usually of rather serious import, patients may live for a considerable number of years even in the presence of this definitely abnormal electrocardiographic finding.

Coronary thrombosis is a frequent complication of angina pectoris and it has been only during the last ten years that a clinical differentiation between the two conditions has been appreciated by the medical profession at large. Mackenzie included eighteen such cases in his series of 160 cases of so-called angina pectoris, nine of which were confirmed by post-mortem examination. Coronary thrombosis occurred in 130 of our total series of 500 cases (about 25 per cent). In this group of 130 patients, it occurred at the onset of the angina pectoris in 40 instances (30 per cent), and was a recognized terminal event in 33 patients (25 per cent). Two attacks of coronary thrombosis occurred in eleven of the patients. Of the 130 patients, 95 are dead (73 per cent) and the average duration of life of those followed from the onset of angina pectoris to death was 4.6 years (the same as for the group of 213 patients followed from the onset of angina pectoris to death). Of the 35 patients still alive after the complication of coronary thrombosis, 22 per cent have angina pectoris only on unusual exertion, 53 per cent have attacks precipitated by moderate exertion and their activities are considerably limited because of angina pectoris, while the remaining 25 per cent have attacks even when quiet and are in a poor condition.

Hypertension was considered to be present in those patients who had, or who had had in the past, a systolic blood pressure of 160 mm. of mercury or more, or a diastolic pressure of 110 mm. or more. There

were 182 of the total series of 500 who presented this finding, an incidence of 36 per cent. Seventy-five of the 182 patients who had hypertension are dead, the average duration of life after the onset of angina pectoris being 4 years, while the total average duration of life including the 107 hypertensive patients still living is 4.5 years. From Table II it is seen that hypertension was present more frequently in the group who succumbed within three years than in the group which survived seven years. It seems that although hypertension may not be of serious import, its presence apparently has a slightly unfavorable influence on the duration of life after the onset of angina pectoris. Our figures do not support a prevalent idea that the presence of hypertension with angina pectoris is a favorable combination.

Gallop rhythm was noted in 29 patients of whom 18 are dead; the average duration of life following the onset of angina pectoris in this latter group was only three years.

Angina pectoris decubitus is usually a sign of considerable gravity and 23 of the patients who had angina pectoris of this severity died within three years, although 16 patients lived more than seven years; thus it is possible but somewhat unusual to survive angina decubitus for many years.

A few further observations of some interest in this series of angina pectoris are to follow. From the etiological point of view, syphilis was present with certainty in only 22 patients, an incidence of 5.5 per cent. A history of rheumatic fever was given by 37 patients, but it apparently did not affect the prognosis. Evidence of arteriosclerosis by physical or by roentgen-ray examination was a frequent finding in this series, occurring in considerably more than half of the patients. The patients were divided into three groups on the basis of the quality of the heart sounds; they were considered good in 52 per cent of the cases with an average duration of life of 5.2 years for this group, fair in 32.7 per cent with an average duration of life of 4.5 years, and poor in 15.3 per cent, the duration of life after the onset of angina pectoris being 4 years. Valvular disease was found in 47 of the patients but apparently did not affect the prognosis, except in those patients who had aortic regurgitation of luetic origin, of whom there were 11 cases; eight of these 11 patients are dead with an average duration of life of 2.6 years, and three are living, the duration of life to date being 2.7 years. Alternation of the pulse was present in 15 patients, of whom 13 are dead.

The relation of angina pectoris to auricular fibrillation is of interest, since it is recognized that the two are rarely present at the same time. Of the total series, this arrhythmia was found in 19 cases. In 11 it occurred in paroxysmal form, and was a terminal event in 4 other cases. In 4 patients both angina pectoris and constant auricular fibrillation were present. Two patients had angina pectoris at the

time of their paroxysms of auricular fibrillation, an unusual and interesting combination, easily confused with acute coronary thrombosis.

One patient with arteriosclerosis and hypertension has had complete auriculoventricular block for 3.5 years with slight congestive failure at intervals and continues to have angina pectoris on exertion, but remains in fair condition.

The severity and radiation of the pain for the group are listed in Table II. Of some interest is the somewhat infrequent radiation found in 8 patients in whom the pain was referred from the substernal region to the epigastrium. In one patient the pain was first felt in the left arm and was later referred to the left chest, while in another patient a typical attack of angina pectoris on exertion began with a feeling of numbness and pain in both hands and lower arms followed shortly by radiation to the chest and upper substernal region. In 20 patients the pain was referred entirely to the right arm, and when present in the right arm the pain appears to be of more serious import than when entirely left-sided.

Attention has already been directed to one particularly favorable prognostic sign and that is the absence of all evidence of heart disease by physical examination, electrocardiography, and roentgen study. The second important favorable prognostic feature is the degree of care taken of the health. There is no doubt in the least that proper attention to the establishment of excellent habits of rest and exercise, diet, and weather, and avoidance of strain of all kinds prolongs life, sometimes for years. Exceptions always exist, but given a relatively young individual, that is under 55 years, who shows no physical evidence of heart disease and who can and will take proper care of himself or herself the prognosis may be considered as probably good for years to come in the absence of angina decubitus.

PROGNOSIS OF CORONARY THROMBOSIS

Although it has been only during the past decade that the clinical picture of coronary thrombosis has become familiar to physicians at large, the combination of a careful history, physical examination, and electrocardiogram often reveals evidence that such an event has occurred in the past, and it has come to be recognized as a common clinical condition. Not infrequently a patient, consulting a physician because of cardiovascular symptoms, dates the beginning of his illness to an acute episode months or even years before, which is readily recognized to have been a coronary occlusion. Much has been written on the diagnosis of coronary thrombosis, so that it is usually a relatively simple matter to make the diagnosis correctly during life providing the thrombosis occurs suddenly enough and in a vessel of sufficient calibre to produce a fair sized infarct in the myocardium, without killing the patient instantly. The prolonged terrible pain

and distress, substernal and epigastric, the failure of nitrites to relieve the pain, the need of large doses of morphine, the prostration for hours or days, and the symptoms and signs of infarction—fever, leucocytosis and sometimes pericarditis and congestive failure—the frequent history of angina pectoris, and the character of the heart sounds and the electrocardiogram, all help in establishing the diagnosis, but it is not essential to find all of these in a given case. To make a correct diagnosis in the absence of pain is more difficult; however, occasionally an acute onset of severe dyspnea (pulmonary edema), congestive failure, or of cardiac arrhythmia, supported by electrocardiographic evidence, may be sufficient to warrant a diagnosis of a painless coronary thrombosis. We have recently seen a case of this type at the Massachusetts General Hospital in which the correct diagnosis was made before death and confirmed several weeks later at post-mortem examination.

There was no clinical doubt of the diagnosis of coronary thrombosis in the two hundred patients of the present series. Two types of patients with coronary occlusion are not included in the present study; namely, (1) those cases in which the occlusion was so sudden that death occurred at once, and (2) those cases in which the occlusion occurred so slowly—by arteriosclerotic narrowing of the vessel, eventually with thrombosis—that no symptoms or signs ever developed except, perhaps, eventual congestive failure. The two groups more or less offset each other prognostically. But when the clinical diagnosis of cardiac infarction is possible, it is of great interest and of considerable importance to know what the chances of life may be. It is perhaps not adequately appreciated that acute myocardial infarction may be, and not infrequently is, compatible with many years of useful and active life, but with a life that should be lived at a somewhat lower level. In this connection a case of unusual interest is included in our series; a man of nervous temperament who had always been a strenuous worker had a classical attack of coronary thrombosis at the age of 63 years, which attack was followed by rest in bed and a prolonged convalescence of several months. Two years later he passed a life insurance examination easily, and nine years later (aged 72) he climbed Mt. Moosilauke in New Hampshire nine times in the course of a year without difficulty. At the present time he is living at the age of 79 years and leads a fairly active life for a man of his age, walking five miles a day, but he has occasional substernal oppression on undue exertion when he forgets himself.

Conner and Holt[3] have recently published an interesting study of the records of 287 cases of coronary thrombosis. They have summarized their findings as follows: "Approximately 85 per cent of these cases were found in men and 15 per cent in women.

"An analysis of the age incidence at the time of the first attack discloses the fact that in one-third of all the cases the first attack occurred before the fifty-first year, and in three-fourths of the cases before the sixty-first year. It seems evident therefore that coronary thrombosis must be regarded as essentially a disease of early middle life rather than of elderly life as it is usually held to be.

"Evidence of an antecedent arterial hypertension was found in 34 per cent of the cases, of syphilis in 14 per cent, and of diabetes in 10 per cent.

"Of the 287 patients studied 117 are known to be living and 142 to have died.

"The immediate mortality in the first attack was 16.2 per cent.

"Of 117 patients who recovered satisfactorily from the first attack, 75 per cent were in good health at the end of one year; 56 per cent at the end of two years; 21 per cent at five years, and 3.4 per cent at ten years. One patient remained in good health for seventeen years and died in a second attack eighteen years after the first.

"In 62 per cent of the patients the first attack supervened, without antecedent circulatory symptoms, in persons who had no reason to doubt the integrity of the heart.

"A single attack only of thrombosis is recorded in 67 per cent of all the patients; two attacks occurred in 24 per cent; three attacks in 4 per cent; and from four to seven attacks in 5 per cent.

"Of the patients having but a single attack, one-half are living and one-third are in good health.

"Among the patients having two or more attacks the time interval between the first and the second attack was less than one year in half the cases and in the other half varied from one to eighteen years.

"Signs of arterial embolism appeared 49 times among 42 patients. Twenty-eight of the embolic attacks involved the systemic arteries and 21 the pulmonary artery.

"Although the immediate mortality in attacks of coronary thrombosis is higher when the initial symptoms are severe than when they are mild, yet almost one-third of the patients who recovered from the attack had symptoms of very severe character."

Levine[4] has published a review of 145 cases in the course of a general survey of coronary thrombosis. As to prognosis he writes:

"There are few diseases in which the prognosis in any individual case is more difficult to predict than in coronary thrombosis. . . . .

"A patient may seem to have had a mild attack and be progressing most satisfactorily, having no complaints and showing nothing remarkable on examination, and then die suddenly on the fifth to seventh day after the attack. The reverse is true, namely that after a very violent attack with serious complication, recovery can take

place. For this reason the prognosis in all cases must be guarded until a few weeks have elapsed, and contrariwise, hope should be held out and every effort in the way of treatment should be carried out in the face of apparently desperate circumstances. . . . .

"The factor of age is of some importance in the prognosis as far as immediate recovery is concerned. The average age of those who recovered was 54.7 years, of those who died 61.0 years, and of the entire group 57.8 years. This would indicate that the younger are a little more apt to recover. It is also true that having recovered from the attack, the younger are more apt to continue in good health for a longer time. . . . .

"The criteria for prognosis in individual cases were found to be most unsatisfactory. In general about 50 per cent have an immediate recovery. No single feature seemed to be reliable as indicative of a good or poor prognosis. Apparently mild cases occasionally died and very severe ones recovered. Slight differences in the mortality were found when certain factors were analyzed such as age, sex, the development of pericarditis, and auricular fibrillation. Ventricular tachycardia and heart-block seemed to have a greater mortality than the average. Even the type of change in the electrocardiogram had no influence on whether the patient would recover or not."

PRESENT SERIES OF TWO HUNDRED CASES OF CORONARY THROMBOSIS

The present series of two hundred cases includes 125 patients with angina pectoris who had coronary thrombosis also, and in addition there were 75 more patients who did not have angina pectoris. The cases have been tabulated, like the cases of angina pectoris, in three groups: (1) total series; (2) thirty-three who, although surviving the acute attack by at least one month, died in less than one year after the attack of coronary thrombosis, and (3) thirty-three who have survived by four years or more the acute attack.

Of the total series of 200 patients (Table I), five have not been heard from recently, and since three of these were in a poor condition when last heard from several years ago, it is probable that these patients are dead. One hundred and one patients (50.5 per cent) are known to be dead, of whom all died cardiac deaths except six, and of these six patients the cause of death could not be determined with any degree of certainty in four, one died of carcinoma, and the remaining patient committed suicide. From this data it will be seen that even though the patient survives the first attack of coronary thrombosis for months or years, he almost invariably succumbs eventually to cardiac failure, either congestive heart failure, angina pectoris, or a subsequent coronary occlusion. It is of interest to compare these findings with those in the series of patients with angina pectoris in which 82 per cent of the completed series of 213 patients

died cardiac deaths. More than one attack of coronary thrombosis is not unusual. Twenty patients of this series survived two attacks, eight patients survived three, and another patient had three definite attacks in the course of several years, but finally succumbed to the fourth.

The average duration of life after the first attack of coronary thrombosis for the 101 patients who died was 1.5 years—ranging from a few hours to eleven years. If we exclude the 21 patients who did not survive the acute attack by one month, the average duration of life of the remaining 80 patients was 1.9 years. The average duration of life after the attack of coronary thrombosis in the 94 patients known to be still alive is 3.2 years, giving an average of 2.4 years for the entire, but uncompleted, series of 200 cases. This figure will be lengthened considerably upon completion of the series when a final report will be made. The longest duration of life following an attack of coronary thrombosis in our series has been 17 years and occurred in a minister who has had two subsequent attacks but at the present time remains in relatively good health at 64 years of age with, however, occasionally substernal oppression on exertion. It has been pointed out before that many of the patients who had the combination of angina pectoris and coronary thrombosis were included in both series. In the preceding study of angina pectoris the complication of coronary thrombosis was found not to have affected appreciably the prognosis. From Table III it is seen that angina pectoris complicated coronary thrombosis an equal number of times (23) in the group which succumbed within one year and in the group which survived four years.

Hypertension was considered present, as in the angina pectoris series, in those patients who had at the time of examination, or who gave a history of having had in the past, a blood pressure of 160 or more mm. of mercury systolic or of 110 or more mm. diastolic. There were fifty patients who presented this finding, and of this group thirty are dead with an average duration of life following coronary thrombosis of 2.4 years, while the remaining twenty who are still alive have had an average duration of life to date of 2.4 years also, which happens to be the same as that of the entire series of 200 cases. It is seen in Table III that hypertension was noted only slightly more frequently in those who died within one year than in those who survived four years. From the data presented it seems that the presence of hypertension in this group of fifty patients has not affected the ultimate prognosis.

Cardiac enlargement occurred twenty-six times in the thirty-three patients who died within one year, and seventeen times in the same number who survived four years. As in the case of the angina pec-

TABLE III

DATA OF TWO HUNDRED CASES OF CORONARY THROMBOSIS

	TOTAL (200 PATIENTS)	DEAD AFTER 1 MONTH AND WITHIN 1 YEAR (33 PATIENTS)	LIVED OVER 4 YEARS (33 PATIENTS)
Sex:			
Male	167 (83.5 per cent)	27	23
Female	33 (16.5 per cent)	6	10
Age at Onset:			
Average	56.7	58	55
Limits	26-80	37-75	39-71
Angina Pectoris:			
Total	125	23	23
Interval between onset and coronary thrombosis			
0-1 year*	73	10	15
1-2 years	9	0	0
2-5 years	29	9	5
More than 5 years	14	4	3
Hypertension (160/110)	50	9	7
Syphilis:			
Positive	7	1	0
Questionable	1	1	0
Negative	192	31	15
Cardiac Enlargement:			
Positive	148	26	17
Negative	52	7	16
Congestive Failure:			
Positive	67	18	5
Negative	133	15	28
Character of heart sounds:			
Good	71	5	11
Fair	69	11	11
Poor	70	17	11
Pericarditis diagnosed clinically at time of attack:			
Positive	20	7	0
Negative	147	20	20
Questionable	33	6	13
Paroxysmal fibrillation at time of attack:			
Positive	13	4	5
Negative	176	28	28
Questionable	11	1	0
Prolonged fever at time of attack	85	13	11
Electrocardiogram:			
Auriculoventricular block			
Positive	11	3	1
Negative	116	12	26
Intraventricular block			
Positive	31	3	11
Negative	96	12	16
Abnormal T-waves (Lead I or II)			
Condition at last report			
Good	68	0	20
Fair	17	0	4
Poor	14	0	2
Dead	101	33	7

*Included in this group are twenty-five patients whose angina pectoris began at the same time as the coronary thrombosis and five patients who had angina pectoris at varying intervals after the coronary thrombosis.

toris series, syphilis was rarely present. Congestive failure was noted in sixty-seven patients and appears to add considerably to the gravity of the prognosis. Forty-six of the patients with this finding are dead and twenty-one are living; it occurred more than three times as often in the group that lived less than a year than in those who survived four years.

A considerable number of this series were not seen by the authors at the time of the acute attack, so that the relative number of those listed as having a pericardial friction rub at that time is undoubtedly much less than their actual occurrence. Pericarditis was noted more frequently in those who died early, while prolonged fever was present only slightly more often in the group that died within one year than in the group that survived four years. The electrocardiogram did not help in determining the gravity of the prognosis. Auriculoventricular block was slightly more frequent in the patients who died early, but intraventricular block as well as an abnormality, usually an inversion, of the T-wave in Leads I or II of the "coronary" type were encountered more often in those who survived. The treatment, consisting primarily of rest with long convalescence and a careful life afterward, was generally much better carried out in the patients who survived than in those who died.

SUMMARY AND CONCLUSIONS

1. A clinical study has been made of five hundred cases of angina pectoris and of two hundred cases of coronary thrombosis. An analysis is presented of certain factors the presence of which may or may not influence the gravity of the prognosis. The importance of the subject is evident because of the increasing frequency of angina pectoris and of the responsible position in the community of many of the patients.

2. Prognosis of Angina Pectoris. Of the series of five hundred patients with angina pectoris two hundred and thirteen are dead with an average duration of life of 4.4 years after the onset of the disease. The average duration of life to date for the total series of five hundred cases is 4.9 years. Of the patients who died death could be attributed to failure of the heart in 82 per cent, and a sudden exitus was frequently encountered. About one-fifth (19 per cent) had normal hearts on physical examination, normal blood pressures, and normal electrocardiograms. This combination of findings was an unusually favorable sign. Coronary thrombosis, although frequently encountered, did not affect appreciably the duration of life, provided the patient survived the acute attack. The presence of hypertension appeared not to be of serious import. The electrocardiogram was of little help in predicting the outcome, although a "coronary" type of T-wave in Lead I or II was seen more often in the patients who died

early. The presence of syphilis, angina decubitus, poor heart sounds, definite cardiac enlargement, congestive failure, or marked arteriosclerosis appears to be unfavorable, and the more of these factors present in the given case, the worse is the prognosis. A vital factor in the prognosis is the degree of care to which the patient can and will submit; the more careful the life of the individual, the longer as a rule will he live.

3. Prognosis of Coronary Thrombosis. Patients often survive coronary thrombosis for many years in good or fair condition. Of the series of two hundred patients with this condition, one hundred and one are dead with an average duration of life following the attack of 1.5 years. The average duration to date for the entire series of two hundred patients is 2.4 years. The patients who died almost invariably succumbed to failure of the heart. The younger patients as a rule lived a little longer; the sex appeared to be unimportant. Neither the previous occurrence of angina pectoris nor its duration prior to the attack of coronary thrombosis has seemed to matter. Hypertension was not important. Syphilis was rarely encountered. Cardiac enlargement of considerable degree is a somewhat unfavorable sign, while the presence of congestive failure and poor heart sounds adds considerably to the gravity of the prognosis. The electrocardiogram did not help appreciably in this series. Good treatment was important.

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THE ELECTROCARDIOGRAM IN LUETIC, ARTERIOSCLEROTIC, AND RHEUMATIC HEART DISEASE*

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THE purpose of this study was to ascertain what electrocardiographic changes would be found in a series of cases of advanced luetic heart disease. In order to have a basis of comparison, it was decided to include like numbers of cases of luetic, arteriosclerotic and rheumatic heart disease. One hundred cases of each group were therefore analyzed. No effort was made to select the cases. The electrocardiographic record book of the Cincinnati General Hospital was taken, and 100 consecutive cases of each group were set down. Thereupon the clinical case histories were gone over, so that the diagnosis could be verified. Cases in which the diagnosis was in doubt were excluded, and next consecutive verified cases were substituted. The cardiovascular material at the Cincinnati General Hospital happens to be very large, so that in going over the records for two and one half years, the 300 cases were obtained without difficulty. All cases showing improvement or remaining unchanged were followed for a period of at least six months. In this study no reference is made to the symptoms and signs presented by the patients. Emphasis is laid chiefly upon the electrocardiographic findings and mortality statistics. It was felt that a comparison of the findings in luetic, arteriosclerotic and rheumatic heart disease would be of value. Analysis of the autopsy findings is reserved for future study. Summaries of the various analyses are herewith presented, with comment.

Sex, Color and Age Groups and General Mortality.—In the luetic group there were 38 deaths. The average age of all the patients was 48.49 years. There were 81 males (63 colored), 19 females. In the arteriosclerotic group there were 28 deaths. The average age was 59.59 years. There were 65 males, 35 females. In the rheumatic group there were 14 deaths. The average age was 30.08 years. There were 56 males, 44 females.

In the luetic group 66 per cent of the cases occurred between the ages of 40 and 59 years. In the arteriosclerotic group 68 per cent of the cases occurred between the ages of 50 and 79 years. In the rheumatic group 70 per cent of the cases occurred between the ages of 10 and 39 years.

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Arrhythmias.—In the luetic group 54 of the 100 cases showed some form of arrhythmia. Of these, 23 died (42.5 per cent). In the arteriosclerotic group 35 of the 100 showed arrhythmia. Of these, 13 died (37.1 per cent). In the rheumatic group 33 of the 100 showed arrhythmia. Of these, 8 died (24.2 per cent). Abnormal T-waves were found in a large proportion of the fatal cases in all groups, in addition to premature contractions. In the luetic group the specific mortality for the cases showing some form of heart-block was approximately 50 per cent. Slightly higher mortality figures for heart-block were found in the arteriosclerotic group. In the rheumatic group the specific mortality was slightly lower.

Fibrillation.—A special study of the incidence of auricular fibrillation in the 300 cases was made. In the luetic group, there were 10 cases, all males, with an average age of 55.6 years. Of these, 4 died (40 per cent). Of the 10 cases, 3 had aortic insufficiency, and 7 had luetic aortitis without insufficiency. All of the deaths (4) occurred in cases of aortitis without insufficiency. The diagnosis was confirmed in each instance at autopsy. These findings are at variance with those of Juster and Pardee,[1] who found fibrillation only in the luetic cases with aortic insufficiency. In the arteriosclerotic group 15 cases (9 males, 6 females) showed fibrillation. The average age was 65 years. Of these, 3 died (20 per cent). In the rheumatic group 18 cases (11 males, 7 females) showed fibrillation. The average age was 43.8 years. Of these, 3 died (16.6 per cent).

Axis Deviation.—In the luetic group 47 cases showed left axis deviation. Twenty-seven of these occurred in patients with aortic insufficiency. Of these, 13 died. Twenty occurred in patients with aortitis without insufficiency. Of these, 7 died. In the arteriosclerotic group 29 cases showed left axis deviation. Of these, 9 died. One case showed right axis deviation. Condition remained unchanged. In the rheumatic group there were 7 instances of left axis deviation. None of these died. There were 9 instances of right axis deviation, with one death.

The incidence of left axis deviation in this series was not as high as in some other series reported. Thus Master[2] found left axis deviation the most common electrocardiographic abnormality in a series of 152 cases of hypertensive heart disease (74 per cent). His second most common finding was a combination of left axis deviation with inversion of T in Lead I. These cases showed a mortality in his series of 27 per cent, while his general mortality was 18 per cent. In the series here reported, this combination occurred seven times in the luetic group. (Mortality 28.5 per cent. General mortality for the group 38 per cent). In the arteriosclerotic group, the combination occurred six times. (Mortality 33 per cent. General group mortality 28 per cent.) Ziskin,[3] in a series of 100 consecutive patients

with hypertension, found left axis deviation in only 44 cases. He made no study of the combination of left axis deviation and inversion of T in Lead I.

In both the luetic and arteriosclerotic groups of the series here reported, the specific mortality for all cases showing left axis deviation was slightly above the general mortality figure, but the difference was not striking.

Lengthened P-R Interval and Heart-Block.—In the luetic group 15 cases showed lengthened P-R interval. Seven of these patients died, showing a specific mortality for lengthened P-R of 46.6 per cent. Fourteen of the luetic cases showed some form of heart-block. Seven of these died, showing a specific mortality for heart-block of 50 per cent. The gross mortality for the luetic group was 38 per cent. In the arteriosclerotic group, 6 cases showed lengthened P-R. (One death. Specific mortality 16.6 per cent.) Five cases showed some form of heart-block. (Four deaths. Specific mortality 80 per cent.) The gross mortality for the arteriosclerotic group was 28 per cent. In the rheumatic group, 12 cases showed lengthened P-R. (Two of these died. Specific mortality 16.6 per cent.) Eight cases showed some form of heart-block. (Three deaths. Specific mortality 37.5 per cent.) The gross mortality for the rheumatic group was 14 per cent.

Abnormal QRS Complex.—Analysis was made on the basis of finding (a) low voltage in all leads, (b) notching or slurring of the complex, and (c) notching *and* widening of QRS (over 0.12″).

With reference to low voltage the figures for all groups were: luetic group 14 cases, 8 deaths (specific mortality 57.1 per cent); arteriosclerotic 13 cases, 5 deaths (specific mortality 38.4 per cent); rheumatic group 11 cases, 2 deaths (specific mortality 18.1 per cent). Hepburn and Jamieson[4] say that low voltage, even though unaccompanied by other electrocardiographic abnormalities is a prognostic sign of serious import. Willius and Killins[5] as the result of their study of a series of cases do not feel that this is a justifiable statement. The subject is still under discussion. In the series here analyzed it is noteworthy that the low voltage cases in the luetic group had a mortality far above the average. In the arteriosclerotic group the excess mortality for low voltage was not nearly so great, while in the rheumatic group it was hardly noticeable. Analysis with reference to notching or slurring of the QRS complex showed no marked increase in the specific mortality in any of the groups. Heimann[6] studied a series of 85 cases of cardiovascular syphilis and referred to a notching of QRS which he found in 30 per cent of these cases. Juster and Pardee in their series of 50 cases of luetic heart disease[1] found only 7 cases showing the notch of Heimann's illustration. (The notch is not accurately described.) In this series of 100 cases, such notching was found only 9 times. Notching and widening of the QRS

TABLE I

ABNORMAL T-WAVE—ALL GROUPS

	LUETIC HEART DISEASE 100 CASES			ARTERIOSCLEROTIC HEART DISEASE 100 CASES			RHEUMATIC HEART DISEASE 100 CASES		
	DIED	IMPROVED	UNCHANGED	DIED	IMPROVED	UNCHANGED	DIED	IMPROVED	UNCHANGED
Inverted I	2	6	0	4	5	0	1	1	0
Inverted II	1	0	0	0	1	0	0	1	0
Inverted I and II	2	4	1	0	6	2	0	1	0
Inverted II and III	1	2	0	0	3	0	0	4	0
Inverted all	0	2	0	3	4	1	2	0	0
Diphasic I	1	1	0	0	3	1	0	0	0
Diphasic II	0	0	0	1	0	0	0	0	0
Diphasic I and II	2	3	0	0	1	0	0	2	0
Isolectric	1	7	1	0	7	0	1	3	0
Low Voltage	2	2	0	1	1	0	0	2	0
Coronary I	6	9	2	3	2	3	2	4	1
Abnormal S-T Seg.	6	5	1	2	9	5	0	2	2
	Coronary T		17 cases	Coronary T		8 cases	Coronary T		7 cases
	Abnormal S-T Seg.		12 cases	Abnormal S-T Seg.		16 cases	Abnormal S-T Seg.		4 cases
	All other abnormal T		41 cases	All other abnormal T		44 cases	All other abnormal T		18 cases
	Total abnormal T		70 cases	Total abnormal T		68 cases	Total abnormal T		29 cases
	Incidence Coronary T		17 (24.2%)	Incidence Coronary T		8 (11.7%)	Incidence Coronary T		7 (24.1%)
	Incidence abnormal S-T		12 (17.1%)	Incidence abnormal S-T		16 (23.5%)	Incidence abnormal S-T		4 (13.7%)

was associated with a specific mortality above the average in each group of this series. This same statement holds good for all three forms of abnormal QRS, considered as an entity in each group.

Abnormal T-wave.—The high incidence of abnormal T-wave in both the luetic and arteriosclerotic groups is in consonance with the findings of many observers. Details of the analysis are shown in the tables. (Tables I and II.)

Attention may be called to the fact that the specific mortality for abnormal T-waves (all forms) is higher in the luetic than in the arteriosclerotic group. So far as the "coronary T-wave" is concerned, the mortality in the luetic group was almost twice that in the arterio-

TABLE II

ABNORMAL T-WAVE—ALL CASES. INCIDENCE AND MORTALITY

LUETIC HEART DISEASE 100 CASES		ARTERIOSCLEROTIC HEART DISEASE 100 CASES		RHEUMATIC HEART DISEASE 100 CASES	
Gross Mortality 38%		Gross Mortality 28%		Gross Mortality 14%	
Cases showing abnormal T-wave (including Coronary T) 70		Cases showing abnormal T-wave (including Coronary T) 68		Cases showing abnormal T-wave (including Coronary T) 29	
Died	24	Died	14	Died	6
Aortic Insufficiency	16				
Aortitis	8				
Improved	41	Improved	42	Improved	20
Aortic Insufficiency	23				
Aortitis	18				
Unchanged	5	Unchanged	12	Unchanged	3
Aortic Insufficiency	1				
Aortitis	4				
Specific Mortality for abnormal T-wave (including Coronary T) 24 (34.2%)		Specific Mortality for abnormal T-wave (including Coronary T) 14 (20.5%)		Specific Mortality for abnormal T-wave (including Coronary T) 6 (20.6%)	
Coronary T-wave (including abnormal S-T Seg.) No. of cases	29	Coronary T-wave (including abnormal S-T Seg.) No. of cases	24	Coronary T-wave (including abnormal S-T Seg.) No. of cases	11
Died	12	Died	5	Died	2
Aortic Insufficiency	7				
Aortitis	5				
Improved	14	Improved	11	Improved	6
Aortic Insufficiency	9				
Aortitis	5				
Unchanged	3	Unchanged	8	Unchanged	3
Aortic Insufficiency	0				
Aortitis	3				
Specific Mortality for Coronary T-wave 12 (41.3%)		Specific Mortality for Coronary T-wave 5 (20.8%)		Specific Mortality for Coronary T-wave 2 (18.1%)	

sclerotic group. Naturally the "coronary T-wave" mortality was lowest in the rheumatic group. Juster and Pardee[1] found the abnormal T-wave in 85 per cent of their 50 luetic cases. The incidence of abnormal T was much higher in their cases of aortitis without valvular lesion. In this series of 100 luetic cases there was an abnormal T-wave in 70 per cent. Analysis of the two groups of luetic cases, simple aortitis, and aortitis with insufficiency, showed that the valvular group had a higher incidence of abnormal T, a higher incidence of "Coronary" T, but the discrepancy as noted by Juster and Pardee was not so marked.

A comparison of all electrocardiographic changes in the two types of luetic lesion showed no findings characteristic for either group.

Normal Electrocardiograms.—In the luetic group there were six normal electrocardiograms, with one death; in the arteriosclerotic group, two normals, with one death, and in the rheumatic group 25 normals, with one death.

SUMMARY

As a result of this study of one hundred cases of advanced cardiovascular lues, using like numbers of cases of arteriosclerotic and rheumatic heart disease for comparison, it seems quite evident that there are *no* electrocardiographic findings pathognomonic of luetic heart disease.

The electrocardiographic picture in luetic and arteriosclerotic disease shows a striking parallelism. The variations from the normal as found are due to the myocardial degeneration, and are not characteristic of the lues per se. While it is true that certain of the arrhythmias and certain abnormalities of the T-wave are of sinister prognostic import, it appears to be quite true that this is the case in the arteriosclerotic as well as in the luetic cases. There is thus no characteristic electrocardiographic picture of cardiovascular lues, and it is not justifiable to make definite forecasts of the prognosis in this disease from the electrocardiogram alone.

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NERVOUS AND MENTAL INFLUENCES IN ANGINA PECTORIS*†

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HEBERDEN[1] described angina pectoris in 1768 and referred to the nervous and mental influences preceding an attack as follows: "In some inveterate cases it has been brought on by the motion of a horse or carriage or even by swallowing, coughing, going to stool, speaking, or any disturbance of the mind." Again he says: "The angina pectoris, so far as I have been able to investigate, belongs to the class of spasmodic, not of inflammatory complaints." In a summary he notes, "the access and recess of the fit is sudden; there are long intervals of perfect health; it is increased by any disturbance of the mind; during the fit the pulse is not quickened; and lastly, its attacks are often after the first sleep, which is a circumstance common to many spasmodic disorders." He sums up and dismisses the treatment very briefly: "With respect to the treatment of this complaint, I have little or nothing to advance . . . . Quiet, warmth, and spirituous liquors help to restore the patients who are nearly exhausted and to dispel the effects of a fit which does not soon go off. Opium taken at bedtime will prevent the attacks at night." He was a pioneer in the study of the complaint, wrote long before the hypodermic syringe and alkaloids came into use, and yet he made many references to the play of nervous and mental influences, the strain of effort in producing an attack, and the benefit of rest, relaxation and sedative remedies. He discovered that opium is useful in the prevention of an attack, an example of therapeutic nervous and mental relaxation.

Ottley[2] in his interesting life of John Hunter, who had angina for 20 years of a life noted for strain, intensity and activity, says, "The spasms about the precordia were frequently reproduced by very slight causes, as trifling bodily exertion or mental irritation. The latter cause was the most frequent, to which the uncontrolled hastiness of his temper rendered him particularly obnoxious; and so sensible was he of the risks to which it exposed him, that he was accustomed to say that 'his life was in the hands of any rascal who chose to annoy and tease him'; a painful thought, that one possessing a mind of such intellectual vigor should, from neglecting earlier to check this infirmity of temper, at length have allowed it 'so to over-master reason'

*Read at the annual meeting of the American Heart Association, Philadelphia, June 1931.

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as to reduce him to hold his life on such a tenure.'' For weeks before his death, he had been in argument and open hostility with his colleagues at the Hospital and he died in a fit of temper after one of them had asked him a question. Even his face is expressive of strain and great intensity of mind. It has been the habit to interpret his angina in terms of his coronary arteries and their pathology. This coronary disease was present doubtless for years, but the frequent anger was an even greater personal pathology because it started the attack. Had he grown graciously old, and cultivated poise and courtesy instead of anger, he would doubtless have lived much longer because his angina would largely have been avoided. There is no proof that, minus high temper and angina, he would not have lived much longer, even with diseased coronaries. The angina killed him and not his cellular pathology. Many more people are afflicted with coronary sclerosis than with angina. Angina occurs without coronary sclerosis. The chest is just the bony cage that encloses the pain save that which trickles out along the nerves.

Heberden wrote very plainly about angina from his own experience and observation. He was uninfluenced by argument, instruments or the opinions of others, and the chief clinical impression made upon him by the complaint was that it belonged to ''the class of spasmodic complaints,'' i.e., sudden, violent, convulsive, exciting, contracting, painful experiences. One may feel that he may die, and yet his pulse not be faster. With the attack he may die. On the contrary, the attack may apparently do no harm. Between attacks one may be as well as ever, only anginous. Eighty different theories exist in an attempt to explain it. ''The whole history of angina from the time of the first observations down to the assemblage of volumes that have appeared in recent years is marked by the same evil—to wit, the vain pursuit of the morbid entity that lies beneath the fugitive phenomena of the syndrome.'' (Gallivardin.[3]) The trend has been to explain ''the morbid entity'' in terms of a cellular pathology. At one time the fad is to explain the pain in terms of too little blood to a tired muscle[4]; at another to say that it is due to tension and strain in an aorta damaged by a vague pathology[5]; and in yet another to say that too little oxygen reaches the heart muscle.[6] White[20] contrasts ''coronary insufficiency'' with ''nervous sensitivity'' as the two essential conditions.

It is well to think of the patient with angina in terms of a personal pathology. This involves a consideration of the ''bad weather'' and ''storm'' periods in the life of the patient as well as the stability or instability of his nervous life. Are the waters of his life calm and deep or wind blown and wave tossed? Nervous and mental influences hinge largely on the emotions, which in turn are related below to the instincts and above to the ways of the intellect. Emotion is hard

enough to describe. It is a fluid and fleeting thing that like the wind comes and goes, one does not know how. All the more difficult it is to describe how the mixture of instinct, emotion and mind,[7] fanned by strain and energy, grieved by disappointment, angered by injury or injustice, or hurried by necessity, may cause something to happen in the chest that comes convulsive and terrible like a veritable epilepsy of the circulation. Civilization as we know it in Western Europe and America, the ambition, effort and community state of mind of these areas, the increasing responsibilities that come with age, and an aging circulation, apparently are the foundations for the increasing prevalence of angina. The inner adjustment to life, the real spiritual control of life, whose outer evidence is a poise and tranquillity of mind, is not very inviting to angina and the anginous life. The strain of life and mind is probably the chief factor in producing angina and the circulatory irritability necessary to this painful complaint.

It is probably as important to realize what is not angina as it is to recognize angina. However much or little the members of the anginal family may be related to each other, each one, as in the human family, is an individual, and its diagnosis, treatment and prognosis is on an individual basis. There are many causes and varieties of thoracic pain, but only one thoracic pain which is angina. A simple grouping of thoracic pain as a working basis is valuable, and permits nervous and mental influences in angina to be more easily evaluated.

1. Angina pectoris is a paroxysmal thoracic complaint not necessarily associated with demonstrable heart or aortic disease, ranging in radiation from the epigastrium to the tips of the fingers and even more distant parts, varying in degree from a simple substernal weight or sensation, through an ache to a tearing pain or a collapsing agony, and associated in the mind with a sense of danger or dying. It occurs usually in the sensitive, nervous type, as the Jew, or in the tense, efficient American, rather than in the dull, happy negro or the calm, accepting Chinaman. There is no characteristic electrocardiogram.

2. The pain of coronary thrombosis, of a location and radiation similar to angina, but of longer duration and associated with fever, leucocytosis and often a pericardial friction rub. Much argument hinges here, but coronary thrombosis is probably a different disease from angina, and the latter is but one of the symptoms of the former. Arterial thrombosis anywhere may give pain. An infected vegetation in the pulmonary artery gives severe substernal pain.[8] Coronary thrombosis has a characteristic electrocardiogram.

3. The pain of acute aortitis, syphilitic or rheumatic in nature as a rule, usually substernal in location and anginal in kind. This pain is probably to be regarded as dependent on infection and a cellular pathology in the aorta, and as one of the symptoms of an acute aortitis.

4. The precordial pain of organic heart disease, valvular disease, and heart failure. It may occur in the big, brave heart of a hypertension before or after actual congestive failure. The pain may be referred rather gently to the left shoulder and arm or appear as a dull ache in the region of the left scapula. It is but a symptom of the underlying disease and failure of the heart.

5. Precordial pains usually transient, but recurring for weeks or months or even years in neurotic patients, more frequently in women. With her mind on her heart and sensitized mentally and emotionally to any thoracic sensation, she easily becomes the "false cardiopath" of the French, and is no rare patient. Some bereaved women sad of mind and weak of body, with precordial pain, the "grief ache" of the English, represent another type. Here the clinician walks cautiously in mind and manner in an effort to be sure of his conclusions, but with no physical evidence of organic disease and particularly if a tachycardia increases during the examination and a dynamic aorta pounds away in the epigastrium, with absent hyperthyroidism and tuberculosis, the whole matter is one for sympathy, psychotherapy and wholesomeness. The location of the pain is rarely substernal, more often apical or precordial.

6. There may be even more difficulty with those cases of neurocirculatory asthenia, the effort syndrome of the English, the "soldier's heart" of our Civil War. Here the evident relation to stress and fear whether in war or peace, the precordial pain, tachycardia, fatigue and even mild cyanosis, make a distinctive picture that needs time, rest and encouragement for recovery.

7. Aches and pains involving the tissues of the left chest wall. Here is a motley group geographically stimulating to a fear of heart disease. Here belong hyperesthesia of the skin, panniculitis of the adipose tissue, fibrositis of the connective tissue, myalgia of the intercostal muscles, intercostal neuralgia, and referred pain from other areas and trauma. "Would you be uneasy if the same pain were on the right side?" is a good question.

8. The pains of the grosser lesions, pericarditis, pleurisy, empyema, aneurysm, mediastinal growths, lung tumors, and referred pains from diseases of the cord and vertebra. These last are often very deliberate in intimating their location and nature.

9. Many exceedingly rare conditions occur in the chest. They surprise the clinician by their very rarity, and yet so much the more need explanation and treatment. A phlebitis of the thoraco-epigastric vein[9] is a case in point. Pain substernal or precordial in location is of particular importance for diagnosis.

Angina is apparently increasing in a community life of increasing strain, and it is well for the clinician to have very terse and clear

ideas about it. As he hears, as he examines, so must he think, separate, evaluate, ascribe and prescribe, and that with a very tactful and reassuring art. One of the many difficulties about angina is that it is so thoroughly subjective in its nature. One goes by what the patient says, and the patient tries to say what he feels. The patient translates his feelings into his words and objective signs of suffering; the clinician translates these words and appearances into his own interpretation. The patient must feel far more than he says to judge by his words, manner and suffering. One patient said to me, "Oh, Doctor, I wish you could see my pain, and then you could measure it and know just how great it is." One carries his emotions, feelings, prejudices, and habits about with him in a distinctively individual way. Notice how individually, proudly one describes "what I eat for breakfast every day in the year." This is probably more true of the educated and cultured than of the uneducated and natural, though to be natural may be to be very cultured. Each individual is an assortment of ways, habits and emotions, and a state of mind as well. Angina is a loose and elusive thing that comes and goes as it wills unless one gets deep into the personality and understands its very fibre and nature. The personality itself rarely understands itself, and needs rather to be understood. The structure and function of the circulation in an adult whose habits of personality are established is not to be separated from the nature of the personality, the gear of the emotional schedule, and the content of the mind. Nervous and mental influences are probably more closely connected with angina[10] than with any other disease of the circulation save possibly the spastic condition called essential hypertension.

Angina breaks into the personality not only with pain but with the threat of danger. A friend, neighbor or relative has "had this 'breast pain' and there was something the matter with his heart, and he died with it." The fear of heart disease, the sense of dying in an attack,[11] the danger of death are added to the cares and burdens of life. Whereas formerly he was safe, now he is unsafe; formerly well, now stricken with pain and that quickly; formerly with life ahead, now death in an attack seems very near; formerly without traffic with those distant beings called "doctors," now he sees one or more of them, needs one of them quickly in an attack, and actually has to take medicine around with him to be safe and ready to ward off the pain and the danger. I have seen a lawyer of most robust body and electric nervous system writhe in bed from attack after attack of angina until he was cowed with fear, three doctors in attendance, relatives weeping, street closed to traffic, and yet relax and soothe himself to peace with a little castor oil daily, for a foul gut, a little bromide and that in a red vehicle for sleep, and a large and gently explanatory dose of psychotherapy, all joined to wholesale faith and hope. The value of the

last two Thayer[12] says no one would exaggerate but a Christian Scientist. The lawyer seems to have lost his angina and is still living.

Both patient and doctor should remember that when death arrives, it has already passed. One who fears death, dies many times before he dies. Moreover, the patient realizes that he is helpless unless the doctor can help him. The doctor may tell him what the disease is, may give him medicines and accent what to do when the attack comes, but after all this is but treating the pain of the attack. It is a hypodermic for pain and nothing more. Only the anginous have angina. To have angina is to be more anginous. Merely to treat the attack is to view the patient bodily, not as a personality, and does not in the least lessen the drive of life that perhaps more than any other factor, causes the angina. It is as Houston so wisely and rebukingly says, to "practice medicine on the veterinary level." But it is another matter to treat the anginous patient for his anginous state, and to view the attack as a flash of lightning in a clouded sky. To be anginous is a state of the nervous system; to have angina is to have nerve pain in the center of the circulation. Increase the tension of the nervous system and its fatigue with a sensitive and aging vascular system in certain people, and the pain comes. We call it angina, the name of a peculiar heart pain. The doctor has much more to do when he treats an anginous patient than when he merely treats angina. He wishes to know what the anginous patient is doing to life as well as what life is doing to the anginous patient. Each person is really busy with two businesses; one the work of his life, his vocation, whereby he earns his living; the other business is just living, the business of his own life, the sum total of himself, his ambitions, endeavors, problems, and particularly their reactions. The purpose of a life is one thing and the process of a life may be quite another thing. Somewhere between the purposes and the processes of a life may be the starting point of many an angina. What the doctor does in the readjustment of the person, which includes all mental and physical processes, actions and reactions, is the real treatment of the anginous state and far more important than only drugs for angina. To treat the anginous state is the real curative and preventive treatment of the angina itself. Though the heart be the location of the attack, the nervous and mental processes may be the source of the anginous state. A case that illustrates these points may but change our clinical focus from the heart to the nervous system.

A gentleman of sixty came complaining of angina pectoris beginning with the first attack on Dec. 24, 1930, and recurring about 12 times since. The pain is substernal, though in the last four attacks it has radiated to the shoulders and down the arms to the elbows, the more painful in the left arm. There is no shortness of breath or sweating, though in the winter cold air seemed at times to bring on an at-

tack. Just stepping into a warm room would instantly stop the pain. In the last four attacks the pain came while he was walking. Recently he had been so worried, he has walked fast, intent on his business and thinking as he walked. He was of average height and build, but one was impressed with his large, fine head, his strong though anxious, worried, and tired face. His wife had telephoned ahead that he had been greatly worried for three years, and to be very gentle with him. He had noticed that an attack might be preceded or accompanied by disturbing gas in the abdomen, or the gas may come alone. The pain is worse when there is much gas.

His father died of angina at 60, though his mother lived to 88. One brother died of alcoholism and one of tuberculosis. His family has been dominant and influential for a hundred years. Many of them occupy influential positions of national importance. As a whole, it is a family of intelligence, efficiency and energy. The trick of power was in the blood and the patient confessed that he had always prided himself in taking the leading part in everything he was in. As a young man he left a small town in another state to come to Atlanta that he might have a larger opportunity to grow rich and prominent. He was a real estate broker, builder, and capitalist. He confessed that he had made and lost three fortunes. The last loss began three years ago, and for this time he has struggled to recoup and retain a little of what he had. He describes his condition quite uniquely, "For three years I have had cumulative worry. I had enough to quit and retire, and then I gritted myself into enough courage to lose. I have been gritting and losing, and now I am reacting from both. I am nervous in the sense of always being tense and straining. I have never learned to go slow and easy. When I played golf, I stepped off the tee first. When I went to church, I was chairman of the board. When I was in a conversation, I led it." There was no conceit here. It was rather the frank statement of his mental make-up. His wife was a little, thin, timid, gentle piece of steel and courage, who said, "You know, Doctor, my husband has never been able to relax and laugh. He is too serious and business-like. He is not playing when he thinks he is playing."

He was showing a lot to a customer on which he had an exclusive sale. He was suddenly told that another agent had sold the lot. He hurried to the City Hall to investigate, was angry at the injustice, and had a most severe attack. It lasted more than an hour and he was sure he was going to die. Neither warm air nor rest aided and he writhed in pain. This made him seek treatment.

Briefly, his physical condition showed one periapical granuloma, a small inactive adenoma of the thyroid, B.P. 150/100, Pulse 74, Temperature 97.5°, slight enlargement of the left ventricle, normal heart sounds, a mild arteriosclerosis, a small stone in the right kidney,

2 plus albumen in the urine, from an old prostatitis, urethritis and cystitis. All reflexes exaggerated. Hemoglobin 70. Electrocardiogram normal except for excess of somatic tremor. Gastric acidity normal. Wassermann test negative. A nervous man, and tense.

What was to be done? Was he to be viewed merely bodily? The prostatitis was very old. The dental granuloma could hardly cause this picture. Was there a common source for these three chief findings, viz., a nervous indigestion of 20 years' standing, a mild hypertension, and a more recent angina? Was the wiser judgment to regard him as a nervous American with the spasmogenic aptitude of the type, a high strung mind and nervous system, the common source for his three chief complaints; their presence the price he had paid for his efforts to lead, to dominate, and to acquire cares and riches? It seemed better to focus on his personality than on his cellular pathology. He said of himself, "My mind leaps ahead and just goes, it gets animated at the least little thing, and I catch myself halfway through an excited conversation and say, look here, this is against doctor's orders, and I must stop and get hold of my self."

It was decided to attack his personality make-up. Only the offending tooth was removed. Everything was explained to him and his wife. He was seen from April 14 to June 22, 1931, always at the office. Little emphasis was laid on the attack and much on him, his nervous system, and his high-gear living. Sodium bromide and chloral-hydrate in half gram doses were given at noon and bedtime daily. Each weekly visit was spent in psychotherapy, which is nothing more than clinical power, presence, and personality flowing through conversation, explanation, and encouragement. His wife aided greatly because the management was her own conviction approved by thirty years of married life. He became a new man in the ways of his mind, worry lessened, the attacks stopped, the blood pressure returned to normal, he slept and looked well, and only transient gas attacks reminded him of his former state. A magnesium salicylate tablet aided the nervous indigestion. The anginal attacks stopped first, then the pressure became normal, then the disturbing and prolonged gas attacks lessened. He lived by the following written directions:

1. Go slow and easy. First of all inside your mind. Second of all with your body. You have led the pack. Now fall in behind. Hurry up slower and be more backward in coming forward. He leadeth me beside very still waters. No yesterday and no tomorrow—just to live until bedtime. Few preferences and fewer prejudices.
2. Take one teaspoonful of the medicine in water at 12 noon, and bedtime, daily.

3. For the present, a warm tub bath, lying quietly in warm water for about twenty minutes, at bedtime. Temperature 90 to 94 degrees—not higher.
4. Reduce your cigarettes one a week for ten weeks beginning with ten.
5. Lie down, shoes off, coat and vest off, collar and tie off, in bed, one hour after lunch, daily.
6. Avoid, as you would a wild beast, hurry, worry, cold, cold winds, overeating, fast eating, emotion and anger. Remember Connie Mack—"Just another ball game."
7. Carry with you the little vial of nitroglycerine. Put one on your tongue and press against the roof of your mouth when the attack begins and repeat every 15 minutes until relieved. Take a tablet when it begins, and sit down. When it is over, go home and lie down.
8. Return once weekly.

This case has been to me a great clinical lesson or rather, there have been many clinical lessons in one case. From the mind and nervous system the trend to spasm apparently involved the heart in the angina, the vessels in the hypertension, and the digestive tract in the nervous indigestion. McCrae's[13] excellent paper, "Angina pectoris; Is it always due to coronary artery disease?" came to my mind. I could not but contrast the two cases of slowly progressive essential closure of both coronary orifices and the ability of these patients to live due to the compensatory circulation furnished by the Thesbian veins, as reported by Leary and Wearn,[14] with the tremendous accent the literature has laid upon the coronaries as the one cause of angina. Levy's[15] essay on angina as "the symptomatic manifestation of many pathological states" is a wise effort to influence the view that there may be many different causes of cardiac pain. I wondered if our nerve resections and injections, except for the relief of agonal angina after all else has been tried, is not really the wrong path for anginal therapeutics. Perhaps angina had given the clinician himself a kind of angor animi about the disease, and he has come to think of angina as a syndrome which has the patient instead of the patient who has angina. Conner's[16] recent contribution in giving graded exercises to patients with angina is a treatment of the anginal state, and an unconscious transfer of the patient's psychology from his attacks to walking, effort, and encouragement. All through McKenzie's book on Angina he touched again and again on the nervous system and the mind, but somehow he never really came near the problem, for he was too imbued with the influence of the coronaries, as Allbutt was of the aorta.

Long ago Osler[17] noted the rarity of angina in the run of hospital patients and in negroes. Arthur A. Stevens recently showing a case in his wards in Philadelphia, remarked, "In this and other hospitals for the indigent true angina pectoris, at least the major form of the disease, is comparatively rare. The case before us is the first I have seen in this hospital for more than a year, although in the same period there have been hundreds of patients with cardiovascular disease in its most extreme forms." Brooks[18] believes the condition is more frequent than in the not remote past, and that it is even increasing in the hospital class, where "it is associated with worry and great stress of life." He well says "that the obligatory demands presented to every person, physical or brain worker, is for more speed, greater production and greater stress in every relation." Morawitz[19] of Leipzig thinks that we should be intensely concerned with angina, for in Germany at least, one has the impression that it is daily on the increase.

Austin Flint[20] in a large practice in a more placid Victorian New York, claims not to have seen a case in five years. When I graduated in medicine thirty years ago, I had never seen a case, whereas now it is one of the common circulatory complaints. White[20] in Boston has seen as many as one hundred new cases in private patients in eight months and three new cases in a single day, which makes him "believe that the situation is appalling and demands some action on our part. Almost certainly the most effective move that we can make is to call a halt on the world's mad rush of today." Of 3,000 patients in New England seen by him, 353 or 11.8 per cent had the complaint. In estimating the value of his experience, it is only fair to say that he devotes himself to diseases of the circulation as a specialty. One wonders if this apparent increase finds its basis merely in a pathology of the coronary arteries. One of my colleagues, L. M. Gaines,[21] has had here in Atlanta, four women, all under concurrent treatment. I knew one of them for many years, and her nervous make-up was identical with the case above described. Such nervous systems could well be described as electric, extravagant, or by the colloquial word, "sparking." They are up and at life by day and night on every occasion and incident.

As hypertension has apparently increased, so has angina. In 1931 in Atlanta, among the better class of whites, particularly over fifty, it is common. Among the negroes subject to every form of cardiac and coronary disease except the influence of nervous and mental strain, it is practically unknown. The negro has far more circulatory disease than the white, added to exposure and the carelessness of a careless race, and no angina. I worked twenty years among sick negroes in both in- and out-patient sections, and never saw a case in a negro. Among approximately 20,000 in-patient negroes in Atlanta, a diagnosis of angina had been made, even by the interns, only five times

in nine years. The staff had never made the diagnosis in a negro. In a study of these five charts bearing the diagnosis, no one is characteristic of the syndrome but rather of coronary thrombosis, acute fibrinous pleurisy, precordial distress from congestive failure and apical distress from religious and musical fervor. Lyons[21] in New Orleans, thinks it may occur occasionally. Musser,[21] in the same city, has never "observed a full fledged, typical attack of angina pectoris in a negro. On the other hand, we frequently see negroes who suffer with cardiac pain but not the definite anginal syndrome." Lemann,[21] in the same city, with an enormous experience and interest in this question, has never seen true angina in a negro, and makes these interesting comments, "I have no explanation for this very striking discrepancy which has engaged our interest and attention for many years. The only probable explanation that comes at once to my mind is the fact that negroes seem to be less highly organized nervously. They seem to bear pain better than do the whites. Has it not also occurred to you that suicide is exceedingly rare, almost unheard of, among negroes? In other words, I am suggesting that while the physical basis for angina and coronary disease is certainly present in negroes, the symptom complex is not present because of the less acute perceptions of pain. I acknowledge that this is not a complete and adequate explanation."

I questioned a very sensible negro about suicide in his race. He said it was not true that the negro did not worry very much. At times there was great worry, but he never worried very long about any one thing at any one time. It was difficult for the negro to worry standing up, and when he sat down he went to sleep so easily that the worry passed away. This discussion concerns only the full blooded negro. Another negro stated that the chief difference between the white man and the negro was that the white man knew how to work but that he did not know how to play. All of this means that the average negro never takes anything very seriously for very long.

W. R. Houston,[21] in Augusta, has never seen angina in a negro apart from the substernal pain in a luetic aortitis, which removes it from our consideration. Stone,[21, 24] of Galveston, writes, "I still have to say that I have yet to see a typical case of angina in a negro." Comparing his cases with those of Wood, Jones and Kimbrough[22] from Virginia and Massachusetts, "It is noted that there are only about one fourth as many cases of angina pectoris among the Texas group as were reported from the two more northerly situated states." George Herrmann,[21] from a large experience, has never seen a case in a negro. J. D. Odum[21] studied the question for me in the Canal Zone and Panama, and found from the records of the Gorgas Hospital, angina listed as a principal disease with five cases among the whites, and two deaths; West Indian negroes, three cases and two

deaths; and not a single case in a negro in the hospital for this year up to May 13, 1931. Whether these cases were essential angina pectoris or dependent upon aortitis or coronary thrombosis was not stated. Among the Indian tribes in Panama, there is no record of a single case. Deaths from angina apparently are nearly twice as frequent in the northern and eastern states as in the southern and midwestern states, and the rarity of angina in the slow-going tropics is well known. The relative rarity in women is notable and suggestive.

The white man, particularly those living lives of stress in urban conditions of competition, work and strain, makes his little plans and lays up cares and riches and takes much thought of the morrow; the negro knows his weekly wage is his fortune, takes each day as it is, takes little or no thought for the morrow, plays, and lives in a state of play, hurries none and worries little. What must it be to live unhurried, unworried, superstitious but not ambitious, full of a childlike faith, satisfied, helpless, plodding, plain, patient, yet living a life of joy and interest? Even the negro child laughs easily, dances, sings, plays, and that usually in rags and wretchedness. Neither does the Chinaman have angina. He has the placid inner life of the Oriental, reinforced by thousands of years of custom and inner peace, and accented by his nature, "this tranquil quietist." Contrast these two sentences from Houston,[10] "Ah yes, the Chinese lack the spasmogenic aptitude—placid, gentle, peaceloving, Buddhist—their ideal, the serene calm of Amida Buddha, with closed lids and folded hands—symbol, the lotus flower scarcely swaying over the still pool." Such is the Chinaman; now for the American. "It is easy to see why the red, hairy, wassail-quaffing, fighting barbarian from the west has the spasmogenic aptitude ready set upon minimal provocation to tighten its coils on him."

In the midst of writing the above in my pleasant, airy room, the phone rang. It rang again, and then again. The Ethiopian maid downstairs very near it, apparently did not hear it, moved not, nor paid it any mind. And why should she hear it? She was in a music land, crooning and singing over her work. Upstairs I was interrupted. Here was a paper, I must finish it, office hours at one—why doesn't she answer it? I tighten with the spasmogenic aptitude, and answer it resentfully. A call from another maid in another home. I tighten another resentful notch, call the maid, upbraid her for her indifference to a sound which by habit, courtesy and necessity I cannot be indifferent to, return to the typewriter, react and smile and try to relax, while the maid went on with her childish talk in ignorant tidbits, utterly oblivious to time, duty and scolding. I try to correct, she accepts. She accepts so habitually that she neither replied to my scolding nor cared to explain. I strain, she lives with the day. She illustrates the advantages of being uneducated, untutored, unambi-

tious, just one who has by nature been born with "an internal adjustment—to change her soul into an attitude of acceptance." But I meet difficulties every day, and many times a day, and try much or little to change and correct the difficulty externally rather than to adjust myself to it internally. The executive said daily to his secretary when he sat down to answer the morning mail, "Bring on the daily grief and let's do the best we can to settle it." It is a habit of life, and finally the nervous system and the circulation can no longer *together* meet the strain, and the patient has angina.

We call a syndrome essential angina pectoris, or essential hypertension, or nervous indigestion, but in reality it can be explained on the basis of the spasm of muscle in different systems dependent not primarily upon an organic or cellular pathology of that system, but rather upon the spasmodic aptitude and constitution of the individual. As is the gear of the nervous system, so is the presence or absence of spasm. "When I look back over the patients I have seen suffering with the benign anginas, it has generally been true that they have had other spasms." (Houston.) A useful illustration that affords rich analogies is congenital pyloric spasm and stenosis in infants, and particularly in the latter. It has as pure a functional pathogenesis as we know, and occurs before individual habits and mental processes can have little, if any, influence. On the other hand, its presence or absence should be indicative of the spasmogenic aptitude characteristic of the race or tribe to which the newborn belongs. Its occurrence may be a measure of the racial gear, and points towards the spasmogenic trend. Among 24,000 Chinese patients analyzed for Houston in The Peking Union Medical College, only one per thousand suffered from evidence of spasm of unstriped muscle. There was only one case of pylorospasm in a premature infant, not one of stenosis, and only eight in adults, that could be called functional. It is to be remembered that the Chinese in their own country do not have either essential angina or essential hypertension. Pyloric stenosis involves smooth muscle, and partakes of the nature of the "spasmodics" as Heberden said of angina. Jacobson's[23] studies afford evidence that "muscular activities are an index of the activities of the nervous system," and this applies to smooth as well as to striped muscle. Further, the tonus of smooth muscle seems to influence striped muscle and vice versa. The tonus of skeletal muscles is increased with the gastric hunger contractions of smooth muscle. Racial susceptibility to the spasmogenic aptitude throws a new light not only on pyloric stenosis, but upon angina, hypertension, nervous indigestion, and other spasms.

The infrequency of pyloric stenosis in the negro as compared with the white child has often been mentioned in pediatric literature. Dr. M. Hines Roberts[21] furnished me with the statistics from three pedi-

atric services in Atlanta. The Grady Hospital receives only white children; the Emory Grady only negro children; and the Eggleston Memorial receives only white children.

Grady Hospital, In-Patient.—5400 white children in 7 years. Pyloric stenosis, 6 cases.

Emory Grady Hospital, Out-Patient.—13,248 negro children in 10 years. Pyloric stenosis, no case. Pyloric spasm, 5 cases, two in infants almost white in color; of the other three only one vomited enough to be classed as a true pylorospasm. Complexion unknown.

In-Patient.—3,439 negro children in 10 years. Pyloric stenosis, no case. Pyloric spasm, no case.

Eggleston Memorial, 2228 white children in 3 years. Pyloric stenosis, 8 cases. Pyloric spasm, 8 cases.

The pediatrician classifies pyloric stenosis as a disease of modern times with a notable increase in the number of cases in the last thirty years. The internist can bear witness to the increase in the number of cases of angina, hypertension, and nervous indigestion among the white races of Western Europe and North America. These two witnesses bear witness to the strange rarity of these three conditions in the negro and the Chinese. The psychology of stress, strain and struggle in the white races is in sharp contrast to the humorous carelessness of the musical negro or the placid acceptance of the gentle Chinaman. The white race talks of speed and records, domination and colonization, drives, rallies, sales and sales-resistance, estates, trusts and results, it increases its effort and income to gratify its desires, and then multiplies its desires. We call it progress and western civilization. The Chinese and the negro accept and, perhaps far more than the white man, conquer both their spirits and their nervous systems. In this sense the white man's burden is his nervous system. We have so far done very little either for angina or hypertension. We have been very gullible about angina. We have viewed it with the credulity of tradition and treated it with the fatuity of empiricism. There is an empiricism in etiology as well as in therapeutics. The conception of the problem as one of racial susceptibility intimates how limited our studies have been. Perhaps when we realize the tremendous power of nervous and mental influences to produce the anginal state as well as the anginal attack, we shall be stimulated to accent the former rather than the latter, and at first at least look for the cause of both in a personality pathology. Then we may accomplish more for the patient. Spasm is a very old-fashioned word. Personality pathology

may take much deep and tactful wading. The patient is far more than a mere body of tissues and organs. The nervous system and the mind, with their trains of emotions and instincts, may give a very real etiology.

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CONTRIBUTORY FACTORS IN CORONARY OCCLUSION*

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IN ALMOST every instance the ultimate cause of occlusion of a coronary artery is sclerosis of the vessel wall;[1, 2, 3, 4] endarteritis obliterans and embolism constituting but rare exceptions to this general rule. Whitten[5] finds that the arterial branches supplying the left ventricle leave the main stem at right angles and pass directly through the musculature, while those to the right ventricle enter the muscle more obliquely. He believes that this different relationship explains the more frequent occurrence of thrombosis in the left ventricle, kinking and consequent vessel wall changes in the main stem being promoted there by the anchoring effect of the branches. This, however, does not explain the presence of sclerosis in the arteries supplying the left ventricle of one patient and its absence in those of another. When the causes of arteriosclerosis in general and of coronary sclerosis in particular have been discovered, obviously those factors which contribute to sclerosis will be recognized as important contributory factors in coronary occlusion, but at present they cannot be evaluated. Whatever its ultimate cause, it is clear that once coronary sclerosis has been established secondary factors frequently contribute to produce thrombosis. The incidence of occlusion does not parallel the extent of coronary sclerosis. Many patients with extensive vessel change never experience clinical occlusion. Occlusion occurs, on the other hand, in individuals showing little evidence of coronary disease.

Careful study of case reports offers, perhaps, the best means of determining precipitating causes and of evaluating their relative importance. Published reports, however, having been concerned particularly with the clinical manifestations and the course of occlusion, too often unfortunately, furnish an incomplete history of the patient and omit details attending the onset of thrombosis. This defect applies also to my own records, so that in reviewing them together with published cases it has been impossible to tabulate completely all data that might bear on the problem. Certain findings, however, appear to be significant.

Relationship to Infection.—The notion has long been widespread that infection in some way predisposes to occlusion. Opinion at

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present is not altogether in agreement upon the matter, but those who report large series of cases find little evidence that infection, either acute or chronic, contributes toward precipitating thrombosis, except in rare instances.[1, 2]

Relationship to Angina Pectoris.—One relationship is at once apparent. Most patients with coronary thrombosis have had attacks of pain conforming more or less typically to angina pectoris. Of Parkinson and Bedford's[1] patients only 38 per cent had had "no previous angina." Levine[2] states that the "great majority of patients had definite angina pectoris antedating the attack." The close etiological relationship between coronary occlusion and angina is further evidenced by the high incidence of occlusion in patients with angina. This is given by White[4] as more than 26 per cent. Even though it may appear that attacks of angina upon occasion may be produced by causes other than arterial,[6] the evidence accumulates that anoxemia of heart muscle resulting from deficient circulation is the basic factor in the vast majority of cases. Evidence becomes increasingly impressive also, that in most instances this deficiency of the circulation results from coronary sclerosis.

So far the relationship between occlusion and angina pectoris is referred to the ultimate cause of the two phenomena rather than to the precipitating causes of occlusion. It is well known that the precipitating causes differ. But this very difference between the factors which precipitate thrombosis in a sclerosed coronary artery and factors which produce a temporary insufficiency of the circulation in such arteries, throws much light upon the mechanism of thrombosis and indicates certain contributory factors in its occurrence.

Factors in the Production of Pain in Angina.—The mechanism by which pain results from inadequacy of coronary flow is not a proper concern of this paper. It must be kept in mind, however, that circulatory insufficiency may be absolute or relative, its duration variable. So far as anoxemia is concerned, transient diminution of blood supply would produce the same result as temporary increase in demand for oxygen. The same result, indeed, would be produced by increase both in demand and in coronary flow provided the increase in blood flow did not keep pace with the demand. Any factor, therefore, which either increases the need of heart muscle for blood or diminishes the coronary flow might produce pain. Anything which increases coronary flow but which at the same time increases more the demand for oxygen might also produce pain.

Factors in the Production of Thrombosis.—Except in cases of embolism, thrombosis ultimately depends upon a lesion in the vessel wall. Possible contributory factors are: (a) a slowing of the circulation and (b) change in the character of the blood. In sclerosed coronary arteries, therefore, any influence which might lessen coronary flow or

anything which might change the blood itself so as to make it more easily coagulable might conceivably precipitate occlusion. Mere increase in need for oxygen unaccompanied by diminution of coronary flow, however, does not favor occlusion, nor does increased oxygen want when accompanied by increased coronary flow; both of which effects may produce angina. Since embolism is a rare cause of coronary occlusion and since lesions of the intima are almost invariably present in patients who experience this accident, study of contributory factors in coronary thrombosis becomes essentially a study of those factors which diminish coronary circulation and of factors which might make the blood more readily coagulable. Review of case reports reveals little evidence suggesting such blood changes. But it does reveal evidence of factors which lessen coronary flow.

Control of the Coronary Circulation.—Anrep and Segall[7] find that in the denervated heart coronary blood flow is regulated entirely by the mean level of arterial pressure, principally by the diastolic level. As diastolic pressure rises coronary flow increases, and vice versa. In the enervated heart, however, change in diastolic pressure, while important, is not the only factor regulating coronary flow. In the heart-lung-brain preparation the flow varies also with variation in cardiac output. It is independent of strength of contraction. This variation in coronary flow with systolic discharge Anrep and Segall attribute to a reflex mechanism through the vagus. When the vagi are cut, volume flow no longer varies with change in output. Stimulation of the peripheral end of either vagus then diminishes coronary flow. These authors reach the conclusion that constrictor fibres are carried by the vagus, dilators by the sympathetics. The rich vagus and sympathetic nerve supply to the coronaries has been beautifully demonstrated by Woolard.[8] Segall[9] and Cruickshank[10] both state that this reflex dilatation of the coronaries with increase in systolic output plays an important part in adapting the heart to the necessity of increased work.

Possible factors in the diminution of coronary flow, then, are the following: (a) lowering of diastolic pressure, (b) lessening of cardiac output, and (c) coronary constriction by vagus stimulation. Any circumstance which might be suspected of contributing toward coronary thrombosis, must be examined particularly with reference to the probability of its producing any of these effects.

Circumstances Attending the Onset of Thrombosis.—The initial evidences of coronary thrombosis rarely appear during unusual effort or excitement, circumstances which characteristically produce angina. Details of onset are given in only 81 cases of Parkinson and Bedford's series. In none was unusual exertion recorded. In a little more than 40 per cent the pain began while the patient was asleep or in bed, "often in the early hours of the morning," and in 32 per cent more

there was no relationship to exertion. In the remaining 27 per cent it began during exertion, but in no instance was the exertion of unusual form.

This almost invariable absence of effort in the onset of thrombosis and its conspicuous association with rest cannot fail to be significant. By far the most common circumstance of onset is during sleep, particularly in the early morning. This is precisely when diastolic pressure is lowest and systolic output smallest. Grollman[11] finds that the blood pressure (both systolic and diastolic) and the cardiac output in sleeping subjects fall progressively until about 4 A. M. Whether this is due to the prolonged rest or to the low phase of a diurnal rhythm, he does not attempt to say.

Lowering of the diastolic pressure as the precipitating cause of occlusion is strongly suggested in the following case:

A woman 45 years of age was examined before operation, not because of any suspicion of abnormality of the circulatory system, but as a routine preliminary. Aside from a blood pressure of 160 mm. systolic and 90 mm. diastolic nothing to attract attention was observed. Just before operation morphine, gr. ¼, was administered, and a few hours later this was repeated. A widespread urticaria rapidly appeared, a result which, it subsequently developed, had previously followed morphine administration. Her blood pressure had fallen conspicuously before the second dose and after that there was a further decline. For seventeen hours frequent readings showed a variation of the diastolic pressure between 30 and 50 mm. A few days later she began to complain of substernal pain. There was a rise of fever, a moderate leucocytosis and a secondary drop in blood pressure. The electrocardiogram showed a deep Q-wave in Lead III with a plateau type of T-wave, features which changed in subsequent records. The interpretation was as follows: Thrombosis of a small coronary artery induced by slow coronary blood flow incident to the prolonged low level of diastolic pressure.

Opposing Factors in Thrombosis and Angina.—Just as lowering the level of diastolic pressure and lessening cardiac output, by diminishing coronary flow, must be regarded as important factors in precipitating thrombosis in the case of an individual with sclerosed coronaries, so in the same individual those influences which raise diastolic pressure and increase systolic output, thus increasing coronary flow, must be considered as lessening the probability of thrombosis. Exertion and excitement are such factors. But while increasing coronary circulation they also increase the need for it, and in individuals with coronary sclerosis the heart muscle may still be inadequately supplied with blood. A patient with coronary disease, therefore, gets angina with exertion, thrombosis while at rest.

It has been noted by many observers that coronary occlusion and thrombosis in other arteries as well, is relatively frequent in patients in the later stages of heart failure. Most authorities believe that systolic output is diminished in this condition. The enforced rest contributes toward the same result. Although it is possible that other fac-

tors promote thrombosis in heart failure, it appears that diminution in coronary flow is a contributory influence.

Vascoconstriction.—The remaining factor which has been shown experimentally to impede coronary circulation is vasoconstriction. Whether or not it is a clinical factor must at present constitute a theoretical problem. If reflex coronary constriction occurs clinically, in cases with impaired coronary circulation the constriction might cause angina, or the coronary lesion might be so extensive that the added factor of constriction somewhere in the arterial tree might be sufficient to precipitate thrombosis. There is much suggestive evidence that it produces these effects. Segall[9] believes that "coronary artery spasm due to central or reflex stimulation of the vasoconstrictor mechanism must be seriously considered and studied as a possible cause of certain types of angina pectoris." This is no new conception. Mackenzie[12] says: "That angina pectoris is the outcome of a stimulation of the central nervous system is so manifest that it needs no comment."

The evidence that the precipitating cause both of angina and of thrombosis frequently is referable to the *gastrointestinal tract* is impressive. In case reports of both, references to indigestion and to the relationship of onset to the preceding meal are conspicuous. Walking, particularly "after a meal," receives special mention by Mackenzie and by McCrea,[6] and by many others as a common cause of angina. Hamman[13] says: "Patients with angina pectoris mostly ascribe their symptoms to indigestion and tell in detail how abdominal distention precipitates the pain, which quickly disappears as gas is eructated." It can hardly be fortuitous that similar references appear in comments upon thrombosis. Levine[2] says that "it frequently occurs during rest, while sitting quietly in a chair *at a dinner table,** or during sleep." Parkinson and Bedford[1] state that "many patients are aroused from sleep by pain, or are seized with it while sitting quietly at rest, *after a meal** . . ." Of the eight cases reported by Werley[14] such references occur in six. In two cases the patient "ate heavily" during the meal before the attack. In another, the attack occurred "just after lunch." Another patient died suddenly "after supper."

None of these authors specifically refers the onset of occlusion in any case to the gastrointestinal tract; yet each mentions digestion or digestive disturbance, and by inference each has been impressed by possible relationship between gastrointestional influences and the development of thrombosis. This suggestion has arisen time and again in my own cases. In many instances the patient has attributed his attack to a particular article of food, to a midnight supper or to long standing indigestion. The matter can hardly be dismissed with the observation that the subjective sensations in occlusion are similar to those produced

*Italics—mine.

by indigestion and that the patient confuses cause and effect. Upon occasion it may be that it is not in the mind of the patient that confusion exists.

If gastrointestinal reflexes upon occasion do lessen coronary flow enough to cause angina or to precipitate occlusion, vasoconstriction must be regarded as the responsible mechanism. Although reflex coronary constriction has been demonstrated, and by the authors quoted above is thought to induce angina; and although evidence is presented in case reports that gastrointestinal reflexes may precipitate both angina and thrombosis in this way, direct proof that coronary constriction may actually result from visceral stimuli appears to be lacking. Other cardiac effects, however, are known to be so produced. That irregularities so arise has long been the conclusion of clinical experience. Pearcy and Howard[15] produced extrasystoles and changes in the ventricular complex by visceral stimulation in dogs, the apparent pathway being via the sympathetics. Hewlett[16] attributes "vagus slowing of the heart

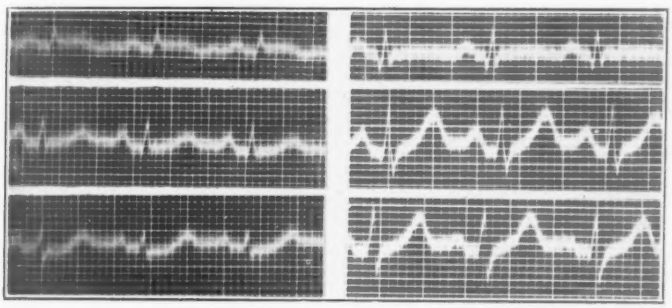

Fig. 1.—Electrocardiograms of the patient in Case 1. At the left is the record made by Dr. Fuller in August, 1929, following the attack. At the right is the record made in November, 1929.

to reflexes that arise from the abdominal viscera." Whatever the reflex path, it would appear that gastrointestinal stimuli as the precipitating cause of coronary occlusion must be carefully considered.

Cold.—Pressure upon the heart through the diaphragm by a distended viscus has long been suspected of producing certain cardiac effects. Pearcy and Howard[15] present evidence that these effects result from reflex stimuli rather than from displacement. Another direct factor, however, must be considered, i.e., *cold.* In two of our cases the evidences of occlusion immediately followed the drinking of cold fluid.

CASE 1. A man 45 years of age, in good health, in August, 1929, was about to start on an automobile trip. He drank several glasses of ice water and began to belch, to "get his breath hard," and to experience a sensation in the chest which he found it hard to describe. Dr. James T. Fuller, who was called, made a diagnosis of coronary occlusion, and treated him on that basis. A part of the electrocardiogram made by Dr. Fuller at the time is shown at the left of Fig. 1. The R-T configuration in Leads I and III is that which characteristically is shown in oc-

clusion involving the apex or anterior part of the left ventricle. In our record of November 29, 1929, shown at the right of Fig. 1, changes have occurred. The patient at the time of the second record was quite well. He had had occasional sensations in the left chest and had noted a few extrasystoles, but has since been in good health.

CASE II. A man of 55 years had always been in good health. Frequent examinations for insurance had disclosed nothing of importance. In July, 1930, while driving in an automobile early one evening he drank a cold beverage and began to experience "heart burn." After about twenty minutes there was severe pain in the upper mid portion of his chest, down both arms and into the neck and right shoulder. There was profuse sweating, dyspnea and slight nausea. When examined a few hours later, he was pale and was still in severe pain. The heart action was rather slow and there was an irregularity which appeared to be of sinus origin. There was no fever. The blood pressure was 140/85 mm. There were no abnormal signs on examination of the lungs and abdomen. Morphine sulphate, gr. ⅜, gave considerable but not quite complete relief, and next morning he still had some pain. The blood pressure then was 110/70 mm. and the mouth temperature was 99.1°. A

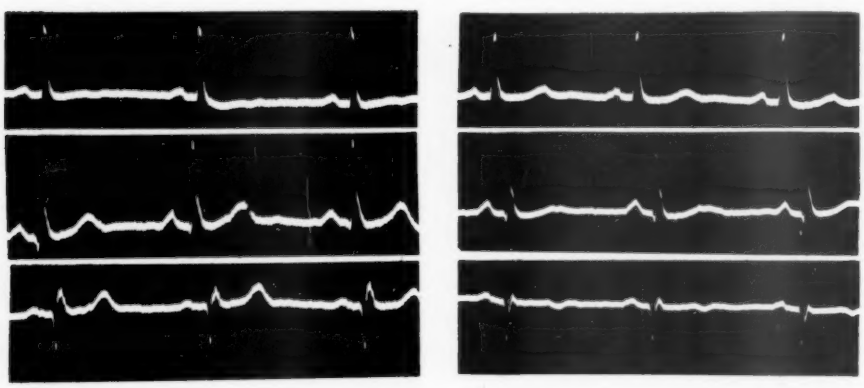

Fig. 2.—Electrocardiograms of the patient in Case 2. The record made one day after the attack is shown at the left. That at the right was made seven days after the attack.

few days later he again experienced some pain for a short time. After that recovery was uninterrupted and except for a feeling of weakness there were no symptoms. Fig. 2 shows the differences in electrocardiograms made one and seven days after the attack respectively. The change in the T-waves of the first and third leads was further accentuated in a later record. The patient has since remained quite well.

It cannot be proved that the occurrence of coronary occlusion with the ingestion of cold fluid in these two cases was other than a coincidence, but in both cases the suspicion of cause and effect must be great. If cold in the stomach precipitated the occlusion, two possibilities must be considered, (a) reflex coronary constriction and (b) the direct effect of cold upon the heart.

The Direct Effect of Cold.—Wilson and Finch[17] observed inversion of the T-waves in Lead III of the electrocardiogram in men after drinking three glasses of iced water. X-ray pictures showed that the in-

ferior aspect of the apex of the left ventricle was in contact with the fundus of the stomach distended by the cold water with only the diaphragm between.

By cooling the apex of the dog's heart, Smith[18] produced a negative T-wave similar to that following ligation of the branches of the left coronary. Otto[19] obtained electrocardiograms showing high branching of the T-wave with inversion of the final part, by cooling the apex of the cat's heart. Anrep and Häusler[20] state that "cooling of the heart muscle, the heat of which is controlled, leads to a diminution of the coronary blood flow."

This experimental evidence lends weight to the suspicion that the ingestion of cold fluid in these two cases precipitated occlusion in a small arterial branch supplying the under surface of the apex.

SUMMARY

Possible contributory factors in coronary thrombosis are: (a) those which change the blood and (b) those which lessen coronary flow. Diminution of coronary flow follows lowering of diastolic blood pressure, decrease in systolic output and constriction of coronary arteries. There is little evidence that change in the blood is often important in causing occlusion. Most instances of occlusion occur under circumstances associated with lowering of diastolic pressure and lessening of systolic output. Evidence suggests that coronary constriction induced by gastrointestinal reflexes may upon occasion play a part in thrombosis. It appears possible that the ingestion of cold fluid into the stomach may at times induce occlusion either by such a reflex or by the direct effect of cold upon the inferior part of the apex of the heart.

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THE FAMILIAL TENDENCY OF CORONARY DISEASE*

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ONE of the interesting features of the recent studies of coronary disease is the growing importance that is attached to the hereditary occurrence of the disorder. In the consideration of the etiology of coronary thrombosis by the writers of a decade or more ago, they either omit any reference to the etiology or else say that the information concerning the disorder is as yet too fragmentary to express an opinion as to the possible factors that are responsible for the disease other than arteriosclerosis. For example, Herrick,[1] in his epoch-making contribution of 20 years ago, says nothing as to etiology, although he does mention the occurrence of arteriosclerosis of the coronary arteries. In a later communication[2] he calls attention to one fact which to our minds is markedly significant; namely, that "the aorta was remarkably free from evidence of sclerotic change. No other lesions were found than the isolated patches of sclerosis in the coronaries." In the two patients who came to autopsy, he notes that these patients had only a short time previously been pronounced in good health. One year later he reported[3] three cases of thrombosis of the coronary arteries, two of which were described in his previous communication, but made no mention of the etiology of the condition. In 1918 Levine and Tranter,[4] discussing the importance of infarction of the heart in the differential diagnosis of acute abdominal conditions, reported upon two cases of coronary occlusion that came to autopsy. In one of these case reports the statement is made in the history that the father of this patient died of angina pectoris, without, however, any further comment upon the etiology of this condition. In 1921, Paullin[5] described four cases of coronary thrombosis, but does not mention the family histories of these patients and does not comment upon the etiology, although one case had been under observation for diabetes mellitus. In 1923 Wearn[6] presented one of the best if not the best study on coronary thrombosis that up to this time had appeared in the English literature. He states that records of these people after a thorough search fail to reveal any factor which might play a direct rôle in the etiology of the condition. Heredity was dismissed at once, for there was only one instance of sudden death in a previous generation. All the patients in the series, incidentally, showed some degree of arteriosclerosis, and it was

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Wearn's belief that those agencies which lead to arteriosclerosis presumably play a more or less indirect rôle in causing cardiac infarction. Thayer[7] pauses in his busy life to reflect on angina pectoris, but does not do more than stress the prognosis and treatment of this particular condition, without mentioning coronary occlusion. Riesman,[8] writing in the same year in the *Medical Clinics of North America*, reports upon the first cases we can find in which there was some attention paid to the familial incidence of the disease. He states, however, "As for the etiology of coronary thrombosis, it coincides with that of coronary sclerosis, and about that we know all too little. Syphilis is not a factor of importance, but tobacco is . . . . ."

Gordinier,[9] in a report of thirteen cases, mentions only one case, Case 3, in which there was any family history of importance. In this particular instance he states that four brothers died of angina pectoris, all of whom apparently died as a result of occlusion of the coronary artery. Christian's article one year later (1925)[10] does not mention the etiology and does not detail case histories in the 70 cases that he reviewed. One feature is of interest in this paper, however, in conjunction with our later discussion of the etiology of the condition, namely that there were 32 patients that were under 60 years of age who died a coronary death. In 1926 Hamman[11] discusses the antecedents of patients who have coronary occlusion. He states that occupation, habits, station in life have no direct bearing upon the condition, but that coronary disease is particularly common to men of intellectual habits and to men who have held positions of responsibility and importance. He rather accentuates the concept that those who are intellectually alert are more keenly sensitive to pain than the so-called lower classes, although the dramatic manifestations of coronary occlusion occur equally often in all.

In 1927 Frothingham[12] gives the details of a case of coronary thrombosis which was of interest because it occurred in a relatively young man who had no previous indications that he was a sufferer from vascular disease. Here we have direct evidence of familial tendency of the disease because in this patient the story goes that his father died at the age of 64 of what was undoubtedly coronary thrombosis, although at the time it was called acute indigestion. Greenberg[13] writes entertainingly of the causation of coronary disease. "Fascinating as it is, to speculate about the cause for things in general, the cause of this disease is no better known than the cause for arteriosclerosis elsewhere in the body. Of the usually mentioned causes, faulty diet, focal infection, alimentary toxic products, metabolic waste products may be dismissed as platitudes. It is possible that hereditary influences play a not unimportant rôle especially under the strain and stress of modern life. Syphilis, diabetes, among the chronic diseases cannot be entirely dismissed as contributing factors. Experiments

with bacterial toxins have yielded no positive results. One must maintain an open mind as to the cause of this disease until better proof is adduced."

It will be observed that the factor of heredity is beginning to be recognized by authors who are reporting upon coronary occlusion. Barker,[14] writing the latter part of this same year, says that undoubtedly there are certain families of certain races who are more subject to coronary thrombosis than others. He calls attention to the fact that the Irish people are relatively free from angina pectoris and coronary occlusion as contrasted with the English, stating that possibly diet, which has been held accountable in the past for this racial difference, may not play as important a rôle as inherited racial predisposition. Although articles appearing in periodicals had begun to stress this factor of familial incidence of the disease, two books on diseases of the heart which came out in 1925 and 1926, respectively, one by Cabot, the other by Vaquez, make no mention of familial incidence of coronary obstruction. Christian[15] in the following year, in discussing the etiology of coronary infarction, mentions that there is often an arteritis of inflammatory nature, sometimes caused by syphilis and at other times by bacterial infection, stating that he believes that arteritis of infectious diseases may play an important causal rôle in cases of infarction as expressed in younger individuals.

The growing appreciation of the important factor of heredity in the production of coronary occlusion is exemplified by conclusion 5 of the summary from Levine and Brown's[16] splendid monograph on coronary thrombosis. They state that hereditary factors, although extremely difficult to analyze, were found to be most important, especially in those patients who have coronary thrombosis at a comparatively young age. Possibly as a part of the hereditary factor there seems to be a certain type of individual who is more apt to develop the disease. They state, furthermore, that "arteriosclerosis is a very rare finding and in some it was limited to the coronary arteries." Also they comment upon the rarity with which syphilis may appear in relationship to the condition and likewise the etiologic insignificance of other infectious diseases.

Wilson[17] wrote last year on angina pectoris and coronary thrombosis as observed in 93 patients. As he expresses the difference between the two conditions, coronary thrombosis may be the most severe expression of angina pectoris. In the etiology, infection seems to play an important part, syphilis not so important, while "a familial tendency may also be in evidence."

Two recent articles in English literature are of exceedingly pertinent interest in relationship to our present theme—that coronary arterial disease exhibits a remarkable familial tendency. Herepath and Perry[18] write that their subsequent report is presented largely with

the hope of eliciting from family practitioners notes of similar cases. In the present instance they report the death of a father at the age of 42, who died suddenly and whose two brothers likewise died sudden deaths—one at the age of 30 and the other at the age of 31, one of whom came to autopsy and was found to have atheroma of the coronary arteries. The third brother died suddenly a month after physical examination. In this case there was the story of anginal pains, although the physical examination, including x-ray of the heart, was normal. Peripheral arteries were not thickened and the blood pressure was 120/70 mm. The electrocardiogram, however, showed the changes of coronary arterial disease. The authors state that the pathological process explaining the death of the patient was a premature senility of the arterial tree, in this particular instance in the coronary arteries, without any evidence of an infective factor. The second paper, by Coombs,[19] has some statements with which we are thoroughly in agreement. "The familial incidence of angina pectoris is well known." He mentions a family of father and four children, all of whom died of coronary disease. In his present series of cases he mentions instances of cardiac infarction in which "there were two pairs of near relatives. One of these pairs consisted of father and son." Particularly striking to us are these statements: "There are two possible interpretations of this familial incidence. One is that it is nothing more than an example of the well-known liability of certain families to arterial degeneration, a liability which is apt to be particular as well as general since it includes a predilection for certain vessels. For example, there are families whose members all die of cerebral hemorrhage. Another suggestion is that aberrations of the coronary arteries which, as Hadfield has pointed out, may throw an undue measure of responsibility on the one trunk, may run in families." Coombs concludes, furthermore, that of the three great infective diseases of the heart, none of them excites the coronary syndrome except extremely rarely. Cardiac infarction, for example, in cardiac syphilis, was observed only once in 88 cases.

The latest book on heart disease, that of Paul D. White,[20] in discussing the etiology of coronary disease and coronary thrombosis, states that faulty metabolism, overwork, high protein diet, infection and heredity are among the many factors suggested, but none has been proved or even consistently found. Farther down in the same paragraph he states that the influence of heredity is supported by the frequent finding of several members of a family with coronary sclerosis.

From the review of articles which have mention in them of the etiology of coronary disease and from the review of other articles which are by men of reputation and authority, it is evident that heredity is coming to be looked upon as a rather important factor in the etiology

of coronary disease. We have called attention to the observation of Coombs, and we are heartily in accord with his conception, that aberrations of the coronary arteries may throw an undue measure of responsibility on the one trunk and that these abnormalities may run in families. We believe that there exist two distinct clinical divisions of coronary occlusion: the one, the occlusion that occurs in old individuals, which is merely part and parcel of the general syndrome of arteriosclerosis and in which those various indefinite, vague and irregular pathogenetic factors operate which are presumably responsible for the arteriosclerosis which exhibits a general sclerosis of all the vessels of the body. On the other hand, there exists a certain group of individuals who die at a relatively early age of coronary occlusion. These individuals do not have hypertensive heart disease or at most have a relatively slight rise in systolic and diastolic blood pressure. They have not had arteriosclerotic heart disease as ordinarily conceived, nor peripheral arteriosclerosis, but they do possess a peculiar liability for degenerative changes in the coronary vessels and they show a peculiar and marked familial tendency to sudden death, which death, of course, occurs presumably as a result of the cardiovascular accident of coronary occlusion. It is in this type of individual, as we have indicated several times in our review of the literature, that this catastrophe occurs when they are under 60 years of age; usually they are people who have always had excellent health, who have never had syphilis or rheumatic fever or other severe infection and who have led active, busy lives.

In substantiation of our conception of this rather sharply defined division of types of arterial occlusion, we would submit a brief account of the family history of Mr. W.:

This patient died in the fifth decade of life. He had always been an active, hard-working, professional man, free from illness, with a blood pressure that averaged 130 to 140 mm. systolic, and who had no evidence whatsoever of peripheral arteriosclerosis. Abrupt death was preceded by about six months of angina-like attacks. Death was relatively sudden and at autopsy the explanation was as obvious as the clinical syndrome; there was a large area of recent infarction in the cardiac musculature. The father of this man died as a result of sudden heart failure. He likewise had been an active, mental and physical worker. He died at the age of 55 years, following a sudden heart attack. His first cousin once removed, who also was a professional man, died when he was slightly past 50 years of age, likewise of what was called then angina pectoris. Death was sudden. Three cousins of the first patient also died in exactly the same way—a sudden death, as far as we can find out in the several instances, with previous anginal attacks which were followed by sudden death. All of these individuals, according to the accounts we can get from present members

of the family, were strong, healthy, active individuals who had very much the same general habitus, slightly overweight, well set up, of short stature, and capable of prolonged and continuous mental and physical effort. These are the definite instances of sudden death that we have been able to obtain in this family history of Mr. W. There is no question as to these facts; but we can add, moreover, that from information from the relatives it is possible that other members of the family may have had a coronary occlusion to cause their death, because two of the immediate forefathers of Mr. W., a grandfather and a granduncle, are said to have died suddenly, but we could find no one who could definitely establish this fact other than through hearsay.

Conclusions.—The thesis has been advanced that there are two quite distinct expressions of coronary occlusion. The one is observed in elderly individuals, possessors of a well marked sclerosis of the arterial tree as a whole and in whom the etiological factors are those of arteriosclerosis in general and represent largely the effects of senescence. The other occurs in men, as a rule, not past the sixth decade of life who do not have generalized arteriosclerosis, who may have relatively slight, but never exaggerated, hypertension, who have been singularly free from past infections and who often give a history of coronary occlusion in several members of their family.

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THE RED CELL SEDIMENTATION TIME IN CORONARY OCCLUSION*

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THE sedimentation time is commonly used as an index of the systematic reaction to infection. Up to the present it has been used in infections of all sorts, but particularly in tuberculosis and in gynecological practice. Recently the rate of red cell sedimentation has been stressed as an index of activity in rheumatic fever.

Löhr[1] studied the sedimentation time as an index of the general body reactions during the healing of noninfected wounds and wound-healing following operative procedures which were not made necessary by infection. He studied also the sedimentation time in uncomplicated fractures. He found the sedimentation time definitely lowered after all operative procedures—clean as well as unclean—as well as after noninfected traumatic injuries; the sedimentation time persisted low for many days. These findings have been corroborated by Heusser[2] who attempts to explain postoperative thrombus formation by blood changes of which the increased sedimentation rate is an index.

Since not only infections, but absorptive necrotic processes due to trauma without infection, cause a lowering of the sedimentation time, we became interested in knowing whether cardiac infarction secondary to coronary vessel occlusion would cause similar changes in the sedimentation rate.

In the majority of patients with an acute occlusion of a coronary vessel, systemic manifestations develop quickly. The most commonly noted are fever and leucocytosis. These findings are stressed in the diagnosis of an acute coronary occlusion. They are, however, of relatively short duration, occurring shortly after the insult and persisting only for a relatively short time.

The sedimentation time has been found pathological in chronic infections even though a normal temperature and blood count existed. It was noted after traumatic and postoperative noninfectious states, that the sedimentation time would remain low for many days after the original insult. Westergren[3] states that the sedimentation time decreases a day or two later than the temperature rise in the infectious states, and remains low for two or three weeks after the temperature, pulse and blood counts have returned to normal.

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These data, showing that the lower sedimentation time appears somewhat later than the general signs of activity and persists for an appreciable time after such signs have abated, made us feel that a rapid sedimentation rate, if present, might be used in the diagnosis of a coronary occlusion in such patients as came to us with a history of an attack at a time when the leucocytosis, temperature and other systemic manifestations had abated.

The test in itself has the value of simplicity and lends itself to ordinary bedside practice.

The literature contains many studies on the sedimentation time in infectious conditions, but nothing could be found on the red blood sedimentation rate in coronary occlusion.

METHOD

The technic used was that described by Linzenmeier[4]—0.2 c.c. of 5 per cent aqueous solution of sodium citrate was placed in a Linzenmeier tube, 6.5 cm. high with a clear diameter of 0.5 cm. This tube was calibrated at 1 c.c.; 6, 12, 18, and 24 mm. Blood was drawn in a dry hypodermic or tuberculin syringe from the median cubital vein and added up to the 1 c.c. mark; the tube was inverted twice, and then held vertically. The time taken for the blood cells to sediment to the 18 mm. mark was noted.

SEDIMENTATION TIME VALUES IN NORMALS AND IN INFECTIOUS DISEASES

Normal values for the sedimentation time by the technic described above have been definitely established. Waugh,[5] Ness[6] and others give the normal sedimentation time to the 24 mm. mark in adult males as 1200 to 1400 minutes; and for adult females 800 to 1000 minutes. Bochner and Wassing[7] give the normal values to the 18 mm. mark as 600 minutes or over. Schmitz and Schmitz[8] report an average sedimentation time in normal adults of about 360 minutes. They suggest that 60 minutes or lower may be regarded as definitely pathological. Baer and Reis[9] feel that a sedimentation time of more than 120 minutes is incompatible with pelvic infection. Polak and Tollefson[10] take 90 minutes as a level, below which they assume the presence of an inflammatory reaction. Numerous other investigators have found figures similar to those given above. One can safely take 60 minutes to 120 minutes as an intermediary zone with values below 60 minutes as definitely abnormal, and values above 120 minutes as not pathological. From the studies of Löhr[1] and Heusser[2] it is seen that while the sedimentation time is not quite as rapid in the noninfectious postoperative or posttraumatic absorptive necrotic processes as in the infections, the same limits of normal may be used.

Since coronary thrombosis is more commonly a disease of advanced years, it is interesting to note the sedimentation time values for

elderly individuals as given by Löw-Beer.[11] This author reports that the sedimentation time increases with advancing years, but that minor infections decrease the sedimentation time more readily in elderly individuals.

While some investigators like Hunt[12] feel that the sedimentation test cannot be relied upon in cases of anemia, others have found that while the sedimentation time is influenced appreciably by variations in the blood cell volume, corrections can be made for such variations. No corrections for cell volume, however, were necessary in this study because anemia was not present in the patients investigated.

Below are presented reports of ten patients who showed the clinical and laboratory data necessary to justify the diagnosis of acute coronary occlusion, in whom the red cell sedimentation rates were studied. No cases are included in this report in which there was any suspicion of the presence of a complicating factor which, like chronic or acute infections, embolic phenomena, etc., might influence the sedimentation rate. Cases I, II, IV, V, VIII and X were uncomplicated. Case III is a diabetic in whom no evidences of infection, either acute or chronic, were noted. Case VI gives a history of having had tuberculosis eighteen years ago. There were no findings indicative of active tuberculosis, either on clinical or on roentgenological examination. Case VII gave a history of having had a gastric ulcer, and having been operated on for a spinal cord tumor eight years previously. Postmortem examination showed a healed calcified pulmonary tuberculosis and a healed gastric ulcer. Case IX had been operated on; an acute appendicitis was not found and fourteen days after the operative procedure, the sedimentation time was still twenty-five minutes. The operative procedure itself, therefore, cannot be the cause of this finding in this case. Cases IV, V and VII were fatal. Cases IV and VII were necropsied and the diagnosis of an acute coronary occlusion and acute myomalacia cordis confirmed.

CASE REPORTS

CASE I.—*M. S.* Male; aged 35 years; truck driver. Admitted to the service of Dr. Rosenthal on November 20th, 1930.

Five days prior to admission, while in the subway, patient was suddenly seized with severe substernal pressure, nausea and vomiting. He was treated by an ambulance surgeon and felt more comfortable in a short time; he still complained, however, of a pain of moderate severity in the left chest anteriorly, and a dry cough. These symptoms persisted to the time of admission. *Family History.*—Negative. *Past History.*—Negative. No cardiac or respiratory symptoms prior to the onset of the present illness. Lues denied.

Examination.—(Five days after the onset of the attack.) Revealed a muscular, markedly hyposensitive adult male, in fair comfort. The only significant findings were cardiac. The heart percussed 12 cm. from midsternal line in the fifth space, had a rate of 70, and a normal rhythm. The first and second heart sounds were of equal intensity, distant, and of poor quality; A-2 was greater than P-2. No

murmurs or pericardial rub heard. No evidence of syphilis found. No evidence of sclerosis in other peripheral vessel fields. Carotid sinus pressure caused a definite cardiac standstill.

The electrocardiogram shows a regular sinus rhythm, ventricular rate of 65; a right axis deviation with a sharp inversion of the T-1 and a high position of the R-T-1. In the second lead the T-wave is isoelectric. The main deflections are all slurred.

The x-ray film suggested a ventricular aneurysm.

All the laboratory data corroborated the clinical impression of a coronary occlusion.

The laboratory data in this case are presented in Table I.

TABLE I

CASE I. M. S.

DAY OF ILLNESS	DATE	MAXIMUM TEMP.	MAXIMUM PULSE	BLOOD PRESSURE S.	BLOOD PRESSURE D.	BLOOD COUNT W.B.C.	BLOOD COUNT POLYS	SEDIMENTATION TIME TO 18 M.M.	REMARKS
4th	11/20/30	101^{2}	70	110	70				Closure on
5	21	101^{4}	86						November 16,
6	22	101^{2}	88	115	70				1930.
7	23	101^{4}	88			17,800	82%	15 min.	Hemoglobin
8	24	101^{6}	68	120	70				90%.
9	25	100	80	95	40	15,600	78%	17 min.	R.B.C.
10	26	99^{6}	80	90	60				5,120,000.
11	27	99^{2}	88			8,200	64%	22 min.	Blood Wasser-
12	28	99^{2}	76	105	70				mann nega-
13	29	99^{2}	96	100	60				tive.
14	30	99^{4}	86						Blood Kahn
15	12/ 1/30	99^{8}	72			7,600	77%	45 min.	negative.
16	2	99^{6}	86						Urine negative.
17	3	99	80	100	65	8,200	66%	60 min.	
18	4	99	80						
19	5	99^{2}	72						
20	6	99^{6}	86	100	65	9,600	71%	60 min.	
21	7	99^{4}	86						
22	8	99^{2}	80	85	50				
23	9	99^{6}	80					80 min.	
24	10	99^{2}	78						
25	11	99^{6}	78	120	80				
26	12	99	70						
27	13	99^{8}	70						
28	14	99	68						
29	15	98^{8}	68						
30	16	98^{8}	70	125	80				
	2/19/31			170	110				
	21		85					150 min.	

CASE II. *A. J. L.* Male; aged 60 years; plumber. Admitted to the service of Dr. Shookhoff on November 13, 1930.

For a year prior to admission the patient had typical attacks of angina of effort, and dizziness. These increased in frequency. The day before admission the patient had a very severe attack of precordial pain radiating to the left arm, which persisted for thirty-six hours and which was not relieved by nitroglycerine or by morphine, gr. ss. Patient had a marked pallor and was covered with a cold perspiration.

Family History.—Wife had two miscarriages and three children died in early childhood; otherwise negative.

Past History.—Lead poisoning with neuritis, thirty years ago. Neuritis of the right arm two years ago. Chronic bronchitis for years. Hypertension for years.

Examination.—Revealed an ashen gray facies and an expression of severe pain. The significant findings were cardiac. The heart percussed enlarged to the left; apical impulse neither seen nor felt; pericardial rub audible; a systolic blow over the aortic area and a loud systolic murmur at the apex, transmitted to the axilla, were heard. The first sound was not audible. Carotid sinus pressure caused a definite slowing of the cardiac rate. Liver palpable two fingers below the costal margin and tender. Congestion râles at the base of both lungs. Definite thickening of the walls of the temporal and radial vessels were noted and eye-ground examination showed a definite sclerosis of the retinal vessels. There were no evidences of disturbed kidney function.

The electrocardiogram shows a regular sinus rhythm, ventricular rate of 100. T inverted in all leads. Left axis deviation. Electrocardiograms taken before this attack show a regular sinus rhythm; left axis deviation; no T- or R-T wave abnormalities.

The laboratory data in this case are presented in Table II.

TABLE II

CASE II. A. J. L.

DAY OF ILLNESS	DATE	MAXIMUM TEMP.	MAXIMUM PULSE	BLOOD PRESSURE S.	BLOOD PRESSURE D.	BLOOD COUNT W.B.C.	BLOOD COUNT POLYS	SEDIMENTATION TIME TO 18 M.M.	REMARKS
	11/11/30			190	100	(Seen at home)			Closure on November 12, 1930.
1st	12								
2	13	100	110	140	105	20,600	90%	100 min.	
3	14	101^{8}	104	135	95				R.B.C. 5,300,000.
4	15	102^{6}	102	110	75				
5	16	100^{8}	98	130	85	13,100	79%	13 min.	Blood Wassermann negative.
6	17	101	94	125	90				
7	18	100^{6}	92	120	80	10,500	77%	18 min.	
8	19	100^{4}	82	130	85				Blood Kahn negative.
9	20	100	88	130	80				
10	21	99	86						Urine negative.
11	22	99^{6}	88	125	80				
12	23	99^{6}	90			12,800	84%	41 min.	
13	24	99^{2}	80	130	70				
14	25	99^{6}	90	125	80			28 min.	
15	26	99^{6-}	90						
16	27	99^{6}	88	130	80	9,600	78%	27 min.	
17	28	99^{6}	88						
18	29	99^{6}	88	120	90	8,300	78%	43 min.	
19	30	99^{8}	78						
20	12/ 1/30	99^{6}	96						
21	2	99^{6}	90	125	90				
22	3	100^{2}	84						
23	4	98^{8}	86					60 min.	
	2/26/31							125 min.	
	4/11/31	(Admitted to Beekman St. Hosp. Mar. 5, 1931.)						48 min.	

NOTE.—Sedimentation Time on Feb. 26, was 125 min. In March, patient had another occlusion; was treated at the Beekman St. Hospital for three weeks, and the sedimentation of 48 min. was taken after he left the Beekman St. Hospital.

CASE III. *J. G.* Male; aged 49 years; clothing merchant. Admitted to the service of Dr. Rabinowitz on April 1, 1931.

Four days prior to admission the patient developed severe pain in the substernal region, the precordium and both shoulders which persisted to the time of admission.

Family History.—Negative.

Past History.—Diabetes mellitus of moderate severity and of several years duration. No previous effort syndrome. Nothing suggestive of lues.

Examination.—Revealed a somewhat dyspneic individual in moderate distress. Heart rate 115, regular. Heart sounds distant, and of poor quality. First sound barely audible. Carotid sinus pressure causes almost a complete standstill. Râles at both bases and the liver edge just barely palpable. Clinical impression—coronary thrombosis.

The electrocardiogram shows a regular sinus rhythm, ventricular rate of 115. The P-R interval is 0.22 seconds. There is a high take-off of the T-wave and a high position of the R-T interval in the first and second leads. T-3 is sharply inverted.

The laboratory data in this case are presented in Table III.

TABLE III

CASE III. J. G.

DAY OF ILLNESS	DATE	MAXIMUM TEMP.	MAXIMUM PULSE	BLOOD PRESSURE S.	BLOOD PRESSURE D.	BLOOD COUNT W.B.C.	BLOOD COUNT POLYS	SEDIMENTATION TIME TO 18 M.M.	REMARKS
4th	4/ 1/31	101	122	95	65	10,500	81%		Closure on
5	2	101^{6}	112	125	70			25 min.	March 28,
6	3	100^{6}	110	100	72				1931.
7	4	100^{4}	98	100	60			18 min.	Hemoglobin
8	5	100^{6}	90	112	78				85%.
9	6	99^{6}	88	120	80			18 min.	R.B.C.
10	7	99^{6}	88	105	65				4,700,000.
11	8	99^{2}	80			11,500	67%		Blood Wasser-
12	9	99^{4}	96	105	63				mann nega-
13	10	99	82	105	60				tive.
14	11	99	80						Blood Kahn
15	12	99^{8}	82						negative.
16	13	99^{2}	80	108	63				Uranalysis:
17	14	99^{6}	78					25 min.	Sugar 6.5%
18	15	99^{2}	88	110	70				to 0
19	16	99^{8}	88	105	65				(4/11/31);
20	17	99	84	110	65				acetone plus;
21	18	99	86						diacetic acid
22	19	98^{6}	86						plus; (nega-
23	20	99^{4}	80	90	60				tive after
24	21	98^{6}	88						4/11/31);
31	28							30 min.	albumin plus;
	5/22/31							85 min.	blood sugar
									300 to 187.

CASE IV. *J. L.* Male; 67 years of age; steward. Admitted to the service of Dr. Alex Louria on January 3, 1931.

Eight months prior to admission the patient was suddenly seized with intense pain in the precordium that radiated down the mesial aspect of both arms to the finger tips; associated with nausea and vomiting. Symptoms lasted forty-eight hours and the patient was in bed for a week. Following this he had transient attacks of precordial pain about every three weeks. The day before admission, while sitting in a chair, the patient had an attack similar to the initial one and vomited six to eight times. Morphine relieved the pain only partly.

Family History.—Negative.

Past History.—Steward on ship to Porto Rico. Had typhoid, malaria. Nothing suggestive of lues.

Examination.—Showed a markedly dyspneic cyanotic elderly male. The important findings were cardiac. The heart percussed enlarged to the left. The apical

impulse was not visible or palpable. Heart sounds barely audible, of poor quality, but regular. A-2 greater than P-2. Marked pericardial friction rub over the apex.

The patient died thirteen days after admission. Autopsy showed a recent thrombosis of the left coronary artery with its resultant myomalacia cordis.

The electrocardiogram shows a regular sinus rhythm, ventricular rate of 95. In leads one and two the R-T interval comes off about the middle of the descending limb of the S. T-wave is very low. All the main deflections are slurred and the QRS intervals are widened. Left axis deviation is present and there are definite evidences of an intraventricular conduction disturbance.

The laboratory data in this case are presented in Table IV.

TABLE IV

CASE IV. J. L.

DAY OF ILLNESS	DATE	MAXIMUM TEMP.	MAXIMUM PULSE	BLOOD PRESSURE S.	BLOOD PRESSURE D.	BLOOD COUNT W.B.C.	BLOOD COUNT POLYS	SEDIMENTATION TIME TO 18 M.M.	REMARKS
2nd	1/3/31	101	110						Closure on January 2, 1931.
3	4	102	128	100	70	17,200	81%		
4	5	102	110					20 min.	
5	6	101	108	94	70				Hemoglobin 80%.
6	7	102	102	100	70				R.B.C. 5,480,000.
7	8	101^{2}	100						Urine negative.
8	9	101^{6}	96	100	70				
9	10	100^{8}	100						
10	11	101^{2}	112						
11	12	102	110	108	70				
12	13	100^{2}	104	100	80				
13	14	100^{6}	106						
14	15	100^{4}	108						
15	16								Died.

CASE V. *A. L. B.* Female; aged 65 years; housewife. Admitted to the service of Dr. Shookhoff on December 3, 1930.

On December 1, 1930, patient got out of bed to close a window, was suddenly seized with weakness, severe precordial pain, nausea and vomiting. These symptoms persisted to the time of admission.

TABLE V

CASE V. A. L. B.

DAY OF ILLNESS	DATE	MAXIMUM TEMP.	MAXIMUM PULSE	BLOOD PRESSURE S.	BLOOD PRESSURE D.	BLOOD COUNT W.B.C.	BLOOD COUNT POLYS	SEDIMENTATION TIME TO 18 M.M.	REMARKS
3rd	12/3/30	101^{2}	120			18,800	90	55 min.	Closure on December 1, 1930.
4	4	100^{4}	122						Died on December 5, 1930.
5	5	99^{6}	130	110	70				Blood Wassermann negative.
									Blood Kahn negative.
									Urine—albumin very faint trace; otherwise negative.

NOTE.—Blood pressure before attack usually 160/100.

Family History.—Father died of a stroke at the age of 64 years. Five children, living and well.

Past History.—Typhoid at the age of sixteen. Treated for hypertension with hypertensive anginal attacks for years. Breast abscess at the age of 56 years. Otherwise negative.

Examination.—Revealed an elderly woman in moderate pain; somewhat anxious; slightly dyspneic; extremely nauseated. The cardiac findings were as follows: rate 120; rhythm regular, with occasional extrasystoles; basilar vessel line widened to the left and left ventricle not enlarged to percussion; sounds of poor quality, especially the first sound which was barely audible; pericardial friction rub heard. No pulmonary signs. No dependent edema. Sclerosis of retinal vessels.

The patient died suddenly on her third day in the hospital. Autopsy consent was not obtained.

The electrocardiogram shows a regular sinus rhythm with a ventricular rate of 120; T-1 inverted. The R-T interval in the second lead comes off from the middle part of the descending limb of the R-2 and the T-wave is inverted. S-3 is the main deflection. All main deflections are slightly slurred and the QRS intervals are widened, indicating a partial intraventricular conduction disturbance. Occasional ventricular extrasystoles are observed. Electrocardiogram taken some time previous to this attack shows a left axis deviation; no abnormal R-T or T-wave changes; occasional extrasystoles.

The laboratory data in this case are presented in Table V.

CASE VI. *A. S.* Male; aged 42 years; gasoline dealer. Admitted to the service of Dr. Shookhoff on April 29, 1931.

Patient was well until April 23, 1931, when he was suddenly seized with dizziness and took to bed. He had vague sternal distress but no real pain. No vomiting. No loss of consciousness. No headache or visual disturbance. On April 28th, patient suddenly became pulseless and very pale. Skin was covered with cold perspiration and he became extremely anxious. Hospitalization was advised.

Family History.—Unimportant; four children, living and well.

TABLE VI

CASE VI. A. S.

DAY OF ILLNESS	DATE	MAXIMUM TEMP.	MAXIMUM PULSE	BLOOD PRESSURE S.	BLOOD PRESSURE D.	BLOOD COUNT W.B.C.	BLOOD COUNT POLYS	SEDIMENTATION TIME TO 18 M.M.	REMARKS
2nd	4/29/31	99²	84	130	80			270 min.	Coronary closure on 4/28/31.
3	30	99⁴	108			16,800	69%		
4	5/ 1/31	99⁸	128					135 min.	Hemoglobin 90%.
5	2	100⁴	116						R.B.C. 4,700,000.
6	3	99⁸	104	120	80	15,800	66%	65 min.	
7	4	99⁴	104	110	70				Blood Wassermann negative.
8	5	99²	96			11,100	55%	60 min.	
9	6	99⁶	80	120	80				Blood Kahn negative.
10	7	99⁴	78						
11	8	99⁶	78			10,700	63%	49 min.	Urine negative.
12	9	99⁶	82						
13	10	99⁶	84						
14	11	99²	86	115	80	10,100	51%	55 min.	
15	12	98⁸	84						
16	13	98⁸	80						
17	14	98⁸	84			9,850	51%	60 min.	
18	15	99²	86						
19	16	98⁸	76						
20	17	99²	80						
21	18	99	82			8,500	58%	90 min.	

Past History.—Worked hard all his life; used tobacco heavily for thirty years. Eighteen years ago had a cough, chills, night sweats; diagnosed as tuberculosis but without positive sputa. No cardiac pain, palpitation or dyspnea on exertion. No edema. Severe occipital headaches for the past three years. Pain in the right foot and in the left shoulder for one year—the latter aggravated by exertion.

Examination.—Showed a markedly hyposensitive individual, appearing slightly anxious and slightly dyspneic. The important findings were cardiac. Apex beat was diffuse, in the sixth interspace, close to the anterior axillary line. The right cardiac border percussed 2 cm. from the right sternal margin. Basilar vessel line widened to the left by percussion. Rate 100; rhythm regular. Sounds of poor quality, especially the first sound. At the apex a friction rub was heard. A loud systolic blow was heard over the pulmonic area and P-2 was accentuated. Râles at both bases and the liver palpable two fingers below costal margin and tender. Carotid sinus pressure causes a distinct slowing of the cardiac rate.

The diagnosis was acute coronary closure.

The electrocardiogram shows an inversion of the T-wave in all three leads with a normal axis deviation. Later on in the course of the disease the T-wave changes disappear and a definite left axis deviation is present.

The laboratory data in this case are presented in Table VI.

NOTE.—This patient had a subsequent attack and was readmitted to the hospital.

CASE VII. *W. B.* Male; aged 60 years. Was admitted to the service of Dr. Shookhoff on February 22, 1931.

Patient had complained of shortness of breath on exertion for many years. Seven weeks prior to admission he was awakened with severe substernal pain and a sensation of choking; nausea and vomiting. He was in bed for two days. Three weeks later he developed pain in the right chest and cough; was in bed for two weeks.

TABLE VII

CASE VII. W. B.

DAY OF ILLNESS	DATE	MAXIMUM TEMP.	MAXIMUM PULSE	BLOOD PRESSURE S.	BLOOD PRESSURE D.	BLOOD COUNT W.B.C.	BLOOD COUNT POLYS	SEDIMENTATION TIME TO 18 M.M.	REMARKS
2nd	2/22/31	101[8]	90	138	90				Last closure on 2/21/31.
3	23	100[8]	108	115	78	10,100	80%	37 min.	Previous one on 2/14/31 (?).
4	24	100	160	90	50				Hemoglobin 80%.
5	25	100	170						R.B.C. 3,850,000.
									Blood Wassermann negative.
									Blood Kahn negative.
									Urine—Albumin faint trace; otherwise negative.
									Died: February 25, 1931.
									Autopsy—showed a left coronary thrombosis.

One week before admission the patient had a second attack of substernal pressure, choking and vomiting; worse than the first attack, and lasting two days; a third attack occurred the day before admission and was the worst of the three.

Family History.—Mother died at the age of 68 years of "apoplexy"; one brother died at the age of 36 years of "Bright's Disease."

Past History.—Heavy smoker. Dry cough for fifteen years; spinal cord tumor removed eight years ago; operated on for perforated gastric ulcer seven years ago.

Examination.—Showed a hyposensitive male in moderate distress; dyspneic; slightly cyanotic. The heart sounds were weak, regular. No adventitious sounds were made out. The lungs showed many basal râles but there was no dependent edema elsewhere. Diagnosis of coronary occlusion was made. The patient died suddenly on his fourth day in the hospital.

Post-mortem examination showed left coronary thrombosis; adhesive pericarditis; myomalacia and myofibrosis cordis with mural thrombosis; healed apical tuberculosis with calcification; healed perforated gastric ulcer.

The electrocardiogram shows a regular sinus rhythm, ventricular rate of 100; T-1 isoelectric; T-2 and T-3 inverted; left axis deviation.

The laboratory data in this case are presented in Table VII.

Case VIII. *M. O.* Male; aged 52 years; grocery clerk. Admitted to the service of Dr. Blatteis on May 14, 1931.

On May 12, 1931, the patient suddenly developed severe precordial pain, chilliness and numbness of both hands. The pain became agonizing, radiated down the inner border of the left arm, and the patient became short of breath. Similar but less severe attacks occurred two and three years ago. Was told he had a hypertension four years ago. Shortness of breath on exertion for many years.

Family History.—Unimportant.

Past History.—Unimportant.

Examination.—Showed a plethoric elderly male with an anxious expression; slight cyanosis and dyspnea. Heart rate 130, regular. Sounds barely audible. Cardiac borders difficult to percuss (chest emphysematous). A-2 greater than P-2. A loud friction rub was audible in the fourth interspace to the left of the sternum. There were coarse râles at the right lung base and the liver percussed slightly enlarged, but there was no pretibial edema.

Table VIII

Case VIII. M. O.

DAY OF ILLNESS	DATE	MAXIMUM TEMP.	MAXIMUM PULSE	BLOOD PRESSURE S.	BLOOD PRESSURE D.	BLOOD COUNT W.B.C.	BLOOD COUNT POLYS	SEDIMENTATION TIME TO 18 M.M.	REMARKS
3rd	5/14/31	102^4	132	128	96	13,400	81%	75 min.	Closure on May 12, 1931.
4	15	103	128	114	88				
5	16	102^4	120	124	98	12,350	87%	16 min.	Hemoglobin 82%.
6	17	102	116	128	96				
7	18	102	100	122	86				R.B.C.
8	19	100^6	100	124	86	16,600	78%	26 min.	4,800,000.
9	20	100^4	100						Blood Wassermann negative.
10	21	100	100	112	74				
11	22	100	98						
12	23	102	114	108	72				Blood Kahn negative.
13	24	100^4	120						
14	25	100	120	106	72	9,400	65%		Urine—Trace of albumin.
15	26	99^8	110	114	76				
16	27	99^8	110	117	80	11,000	75%		
17	28	99^4	100	98	66			50 min.	
24	6/ 4/31							120 min.	

The electrocardiogram shows definite evidences of an acute myocardial involvement.

On May 26, patient again experienced an attack of precordial pain. On auscultation a friction rub again was heard. He is still in the hospital at this writing but is free of symptoms. On June 4, 1930, the sedimentation time was 120 minutes.

The laboratory findings are presented in Table VIII.

CASE IX. *W. S.* Male; aged 45 years; clothing operator. Admitted to the surgical service on May 5, 1931.

Two days before admission he developed severe umbilical cramps that radiated to the back; associated with nausea, but no vomiting. Temperature 100-101. No right lower quadrant pain. Symptoms persisted to the time of admission.

Family History.—Negative.

Past History.—Negative. Venereal disease denied.

Examination.—Showed a plethoric adult male, with a flushed facies. Patient was of the hyposensitive type. No dyspnea or cyanosis. Pupils regular, equal and reactive. Throat injected moderately. Lungs negative. Heart—rate regular, 100. No enlargement. Sounds of good quality; A-2 greater than P-2. No murmurs. Abdomen—Negative. No definite tenderness, rebound tenderness or rigidity. Extremities negative.

The diagnosis of acute appendicitis was made and appendectomy performed on May 6, 1931. At operation the appendix did not look inflamed. The pathological report was chronic catarrhal appendicitis. The next day the patient developed a rapid, irregular pulse and an electrocardiogram was taken.

The electrocardiogram shows an auricular flutter with a ventricular rate of 188 and a 2:1 block.

The clinical course and electrocardiographic findings made the probable diagnosis coronary thrombosis.

Electrocardiogram taken on May 24, shows that the auricular tachycardia is no longer present; a regular sinus rhythm with a ventricular rate of 97 is now present. The P-R interval in the second lead is 0.26 seconds (partial A-V block) and there is a left axis deviation. Definite evidence of myocardial damage is present.

The laboratory findings are presented in Table IX.

TABLE IX

CASE IX. W. S.

DAY OF ILLNESS	DATE	MAXIMUM TEMP.	MAXIMUM PULSE	BLOOD PRESSURE S.	BLOOD PRESSURE D.	BLOOD COUNT W.B.C.	BLOOD COUNT POLYS	SEDIMENTATION TIME TO 18 M.M.	REMARKS
3rd	5/ 5/31	101^{8}	120	112	80	14,200	81%		May 6, 1931, appendectomy.
4	6	102^{4}	126						
5	7	102^{8}	130	146	82				Closure on May 3, 1931 (?).
6	8	102^{4}	120						
7	9	101^{8}	104						Urine—Negative.
8	10	100^{4}	100						
9	11	100^{6}	88						
10	12	101^{8}	100						
11	13	102	92						
12	14	102	92			9,000	69%		
13	15	101^{6}	100						
14	16	102^{4}	84						
15	17	102^{4}	80						
16	18	102^{8}	66						
17	19	102	72	120	80			25 min.	

NOTE.—Temperature returned to normal on May 27. The sedimentation rate at that time was 65 minutes.

CASE X. *L. A.* Aged 63 years. Patient of Dr. Shookhoff.

This patient has had a hypertension for many years. One year ago he had an attack of pain in the chest with radiation down the arms and a diagnosis of a coronary occlusion was then made.

Family History.—Father and mother died of unknown cause. Had one brother who had a hypertension and coronary disease. Family and past history otherwise negative.

Patient had been moderately well, experiencing no symptoms up to the onset of this present attack, when on April 30, 1931, he developed a severe pain in the chest, radiating to the back and down both arms—pain was vise-like, excruciating, and required two half-grains of morphine sulphate to relieve him.

Examination.—Revealed an elderly man, brownish greyish facies and slightly cyanosed. Blood pressure 100/80 mm. Many congestive râles at both bases. Heart—aortic configuration, left ventricular enlargement. Heart sounds were distant; first sound was barely audible; carotid sinus pressure caused a complete cessation of cardiac activity for a short time. The cardiac rate was about 120. The abdomen was very slightly distended; liver was palpable two fingers below the costal margin and tender. No edema.

One day after the attack, April 31, 1931, the leucocyte count was 12,500 white cells with 70 per cent Polys., and the sedimentation time was 170 minutes.

This patient was treated at home and could not be followed properly. However, a sedimentation time taken on May 6 (six days after onset of attack), was 30 minutes.

He subsequently developed a cerebral insult causing a left hemiplegia and no further studies were made.

RESULTS

The sedimentation times ranged from 270 minutes to 13 minutes. Sixty minutes was taken arbitrarily as the point below which the sedimentation time was interpreted as definitely pathological. The value fell below sixty minutes in all the cases.

In Cases II, VI, VIII and X, the first sedimentation rate estimations were made on the day following the attack and, while the temperature and blood picture were already abnormal, the sedimentation readings were still normal. They became more rapid subsequently. In Cases I, III, IV, V and IX, the first sedimentation test was not made until several days after the attack and the figures were all below normal. In Case VII the sedimentation time of 37 minutes was obtained on the third day of illness, but there had been recent previous attacks. These cases seem to show that the sedimentation time becomes pathological later in the disease than the temperature and blood count. The lowest level was reached as early as the fifth day following the closure in Cases II and VIII, and as late as the eleventh day in Case VI. Thereafter, in all cases the values increased toward normal. Tables I, II, III, VI, VIII and IX show that the low sedimentation time outlasted, by an appreciable number of days the return of the temperature and blood count to normal. Only one sedimentation time each was obtained in Cases IV, V and VII; these patients died. The sedimentation times in these latter three cases were

all low. The results indicate that the sedimentation time is definitely shortened in cases of acute coronary occlusion. The cases presented in this study had no known infections or other complications that might have caused the abnormal sedimentation times. The more rapid rate occurs relatively late in the disease; it persists, however, longer than does the fever and leucocytosis. This determination, therefore, may be valuable in cases appearing for examination a number of days after an attack and presenting a normal temperature and blood count.

From this study we are unable to state whether or not the extent of the drop in the sedimentation rate is proportional to the extent of tissue destruction in the heart. In studies on the sedimentation rate in other conditions, a relationship between the rate of sedimentation time and extent and severity of lesion has been sought. Thus, in a study of 250 gynecological cases, Benischek and Douglas[13] concluded that the speed of sedimentation was directly proportional to the extent and severity of the pelvic inflammation. Tappan and Faget[14] investigated 234 cases with pulmonary tuberculosis and found the sedimentation rate to be proportional to the extent of anatomical involvement and to be an index of activity. The same relationship between extent of lesion and sedimentation rate may be found in coronary occlusion when a large enough number of cases have been studied and checked up by post-mortem findings.

It seems justifiable to assume that while the sedimentation time is abnormally low, active changes are taking place in the heart muscle. We feel that this test offers a valuable index of the repair process and that the infarctive reaction cannot be regarded as having subsided until the sedimentation rate has risen to at least above the sixty minute level. Polak and Tollefson[10] state that when the sedimentation time is rapid in gynecological cases, they have always found, by operation or by further observation of the clinical course, existing pathological conditions to account for the low determination.

The rapidity with which the value returns to normal is in all probability related to the speed of healing. For this reason repeated determinations of the sedimentation rate may aid materially in prognosis and treatment. In the light of the results presented even in this small series of cases, it appears advisable to keep a patient with a coronary occlusion in bed for at least as long as the sedimentation time still suggests active myocardial changes, i.e., as long as it is below sixty minutes.

We hope that this brief report will encourage further study of the sedimentation time in this type of disease, with a view to establishing this simple procedure as an aid to diagnosis, prognosis and treatment of acute coronary occlusion.

SUMMARY AND CONCLUSIONS

The red cell sedimentation time was studied in ten patients who presented sufficient clinical symptoms and laboratory data to make probable the diagnosis of an acute coronary occlusion. Two of three patients who died were necropsied and coronary occlusion with an acute myomalacia was found.

It was observed in these cases that the sedimentation time was definitely shortened, that this change appeared later in the disease than did the fever and leucocytosis, and persisted for some time after the temperature and blood count had returned to normal. The persistence of this rapid sedimentation rate beyond the time when the temperature and blood changes had disappeared made it, for us, a better index of the progress of the healing of an acute myomalacia due to a coronary occlusion than the temperature and leucocytosis.

NOTE: We wish to thank Drs. Rosenthal, Rabinowitz, Louria, and Blatteis for their kind cooperation in allowing us to use their clinical material.

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CARDIOVASCULAR DISEASE

A Report of 398 Cases Coming to Necropsy*

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IN THE four years from 1927 to 1930, out of 2344 autopsies at the Cincinnati General Hospital the primary cause of death in 398, or 17 per cent, of the cases was breakdown of the cardiovascular system. In this group of "cardiovascular deaths" all cerebral vascular accidents are included but arteriosclerotic kidney cases are omitted because the connection between the renal lesion and the death of the patient is often indefinite. In all of these cases the diagnoses have been carefully controlled by microscopic examination and so possess more value than would the clinical, or even the gross pathological diagnoses alone. These 398 cases have been analyzed in regard to (1) type of cardiovascular disease (classification of the American Heart Association), (2) inactivity period before death, (3) age at death, (4) race, (5) cardiac hypertrophy.

The percentage of autopsies in which cardiovascular breakdown was the primary cause of death varied little from year to year during the period. In 1927 it was sixteen; 1928, nineteen; 1929, seventeen; 1930, sixteen.

TYPE OF CARDIOVASCULAR DISEASE

As to the type of cardiovascular disease at death the etiological classification of the American Heart Association has been followed, with the omission of the diagnoses: thyroid, toxic, neurosis (cardiac) and trauma. In the beginning of this study the syphilitic and the arteriosclerotic heart cases were entirely separated, but this was not found to be practical since a large number of those cases in which death was primarily due to syphilitic heart disease, also showed minor evidence of arteriosclerosis, and some cases in which death was primarily due to arteriosclerotic heart disease, also presented minor evidence of syphilis. For this reason it was necessary to make certain changes in classification. Of the 398 cardiovascular deaths, 164 or 41 per cent resulted from arteriosclerotic heart disease, and 50 or 13 per cent from arteriosclerotic heart disease with minor evidence of syphilis. Combining these two figures there were 214 deaths or 54 per cent caused by arteriosclerotic heart disease. Fifty-five deaths, or 14 per cent of the 398 cardiovascular

*From the Department of Pathology, University of Cincinnati, and Cincinnati General Hospital.

deaths, resulted from syphilitic heart disease, while 34, or 9 per cent resulted from syphilitic heart disease with minor evidence of arteriosclerosis. Combining these last two figures there were 89, or 23 per cent, which were caused by syphilitic heart disease. The remainder of the 398 deaths are classified as follows: rheumatic heart disease 11, or 3 per cent; acute and subacute bacterial endocarditis 36, or 9 per cent; unclassified 8, or 2 per cent; congenital heart anomalies 14, or 4 per cent, and pericarditis, acute and chronic 13, or 3 per cent. In the last group it was necessary to depart from the etiological classification and pass over to an anatomical classification. (See Table I.)

TABLE I

TYPES OF CARDIOVASCULAR DISEASE WITH PERCENTAGES

A. Arteriosclerotic heart disease	41%
B. Arteriosclerotic heart disease + syphilitic heart disease	13%
C. Syphilitic heart disease	14%
D. Syphilitic heart disease + arteriosclerotic heart disease	9%
E. Rheumatic heart disease	3%
F. Acute and subacute bacterial endocarditis	9%
G. Unclassified	2%
H. Congenital heart anomalies	4%
I. Acute and chronic pericarditis.	3%

Of the arteriosclerotic cardiovascular deaths, 65, or 30 per cent, were due to cerebral vascular accidents, while only 12, or 13 per cent, of the syphilitic cardiovascular deaths resulted from such accidents.

In this entire series of cardiovascular deaths, 35 per cent of the cases showed some evidence of syphilis, while only 13 per cent of routine autopsies at the Cincinnati General Hospital, over the same period of time, presented evidence of syphilis.

PHYSICAL INACTIVITY PERIOD FOR VARIOUS TYPES OF HEART DISEASE

By the phrase "Physical Inactivity Period" is meant the length of time that elapses between the onset of cardiovascular symptoms of such severity that the patient is no longer able to maintain his normal activity and the time of death. Here the cases were divided into the same etiological groups as before. For the arteriosclerotic group the inactivity period averaged 10.1 months, while for the arteriosclerotic cases with minor evidence of syphilis it averaged 14.0 months. The inactivity period average for the entire arteriosclerotic group was 11.3 months. From the above figures it appears that the persons with arteriosclerosis and syphilis have a longer inactivity period than those with arteriosclerosis alone. The reason for this is that in the arteriosclerotic cases with minor evidence of syphilis there were a few patients that lived over seven years, and these few were sufficient to raise the average inactivity period for this group. The patients with syphilitic heart disease had an inactivity period of 6.4 months, while those with syphilitic heart disease

with minor evidence of arteriosclerosis had an inactivity period of 12.8 months. The inactivity period average for the entire syphilitic group was only 8.7 months, as compared with 11.3 months for the arteriosclerotic group. In the syphilitic heart disease group the same paradox appears as in the arteriosclerotic group, and the same explanation holds true here, only the syphilitic heart disease group was smaller so the difference caused by a few cases living over seven years was even greater. The inactivity period for the rheumatic heart disease group was 6.6 months while for the acute and subacute bacterial endocarditis group it was only 2.5 months. The inactivity period for the pericarditis heart disease group as well as the congenital heart anomaly group was not determined. The brevity of the inactivity period for these cases may appear discouraging, even alarming, but it is neces-

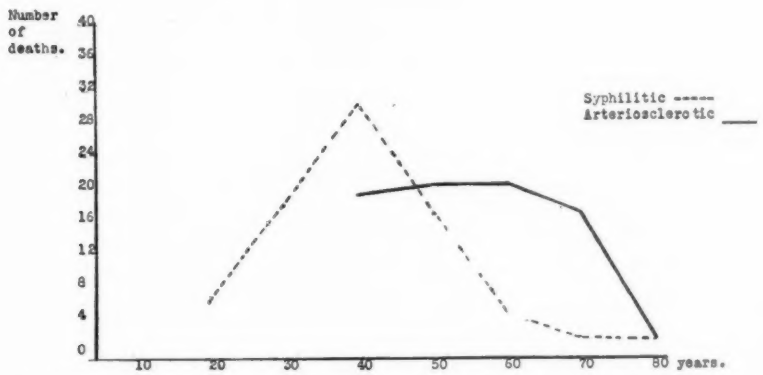

Fig. 1.—Age at death in 80 arteriosclerotic and 80 syphilitic cardiovascular deaths.

sary to make note of the fact that the patients represented here came largely from the indigent and underprivileged ranks of society, and included a number of negroes.

AGE AT DEATH

The average age at death of the patients dying of arteriosclerotic disease was 59.4 years, while of those dying of arteriosclerotic disease but also showing minor evidence of syphilis it was 57 years. Combining these two figures this represents an average of 59.1 years for the entire arteriosclerotic heart disease group. The average age at death of the patients dying of syphilitic heart disease was 45 years, while of those dying of syphilitic heart disease with minor evidence of arteriosclerosis it was 46.6 years. This represents an average of 45.6 years for the entire syphilitic group as compared with 59.1 for the entire arteriosclerotic group. The average age at death for the rheumatic heart disease group was 26.5 years, while that for the bacterial heart disease group was 38.4 years. In the remaining types the number of cases was not considered large enough to warrant averaging the age at death.

In 80 unselected cases of arteriosclerotic cardiovascular deaths and a similar number of syphilitic cardiovascular deaths, the age at death was plotted against the number of cases dying at that age. The results are striking. In the syphilitic heart disease group there is a very definite peak at 40 years and then a rapid decline, while in the arteriosclerotic heart disease group there is a rather broad plateau extending over the fifth, sixth and seventh decades. (See Fig. 1.)

RACE

In the routine autopsies at the Cincinnati General Hospital from 1927 to 1930 56 per cent of the patients were white and 44 per cent black. In the series of patients dying of arteriosclerotic cardiovascular disease, 38 per cent were black and 62 per cent were white, while of those dying of syphilitic cardiovascular disease, 80 per cent were black and only 20 per cent were white. The above figures suggest that syphilitic cardiovascular disease is approximately four times as common in the black as in the white race.

CARDIAC HYPERTROPHY

An attempt was made to appraise some of the factors causing cardiac hypertrophy. In the arteriosclerotic heart disease group there was no constant correlation between the amount of hypertrophy and the condition of the macroscopic coronary vessels, but there was a fairly constant correlation between the length of the physical inactivity period and amount of hypertrophy; in other words, the longer the inactivity the greater the hypertrophy regardless of the condition of the macroscopic coronaries. In the syphilitic cardiovascular group the average heart weight in cases with aneurysms was less than the average heart weight in those cases without aneurysms; that is, aneurysms as such do not lead to cardiac hypertrophy. The heart weight in those cases with adhesive pericarditis was greater than that in any other group.

SUMMARY

A statistical study, of 398 autopsied cases dying primarily of a breakdown of the cardiovascular system, is presented, concerning type of cardiovascular disease, physical inactivity period before death, age at death, race and cardiac hypertrophy.

VENTRICULAR PAROXYSMAL TACHYCARDIA: REPORT OF A CASE IN A PREGNANT GIRL OF SIXTEEN YEARS WITH AN APPARENTLY NORMAL HEART*

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WE ARE reporting this single case of ventricular tachycardia because of the several unusual and interesting features which it presented. These were: (1) the youth of the patient (sixteen years); (2) the almost continuous presence of the disturbance; (3) the possible relationship of pregnancy; (4) its improvement under digitalis and its abolition by quinidine; (5) the absence of any demonstrable cardiac disease; and (6) the fact that cesarean section was performed uneventfully during this disturbance of the cardiac action.

CASE REPORT

The patient, a young, colored girl of sixteen, illegitimately pregnant, came to the Pre-natal Clinic of the Philadelphia General Hospital in the seventh month of gestation. At this clinic, a rapid and somewhat irregular heart action was discovered, and she was referred to us for study.

There were no cardiovascular symptoms whatsoever, not even consciousness of the paroxysms of rapid heart action. Nor were there any symptoms referable to the gastrointestinal, genitourinary, respiratory, or nervous systems. The family history was entirely negative. The past medical history, likewise, revealed nothing of importance. There had been infrequent, mild sore throats, but nothing further suggestive of rheumatic fever; she was not even aware of having had any of the ordinary diseases of childhood.

The physical examination was largely negative. Except for being under normal height (she was five feet tall and weighed 102 pounds) she appeared normally developed and nourished. The head, teeth and tonsils revealed nothing abnormal. There was slight fullness in the region of the thyroid gland. The lungs were entirely normal, both clinically and by x-ray.

The heart was not definitely enlarged by percussion. The left border measured 10.0 cm. to the left of the midsternal line in the fifth interspace and the right 2.5 cm. in the fourth interspace; the retrosternal dullness in the second interspace was 5.0 cm. By teleroentgenogram, the total diameter of the heart measured only 11.2 cm. with a maximum intrathoracic diameter of 22.8 cm. The heart sounds were not remarkable and there were no murmurs. On our first examination, the heart was found most of the time to be beating regularly at a rate of 150 per minute with an occasional brief period of a slower rate. The mechanism was shown by electrocardiograph to be ventricular paroxysmal tachycardia with brief intervals of sinus rhythm interspersed. The blood pressure was 130/80 mm. The abdomen showed nothing of note except the enlarged uterus, and the extremities were not abnormal.

*From the Division of Cardiology of the Philadelphia General Hospital and The Robinette Foundation for the Study of Cardiovascular Diseases of the University of Pennsylvania.

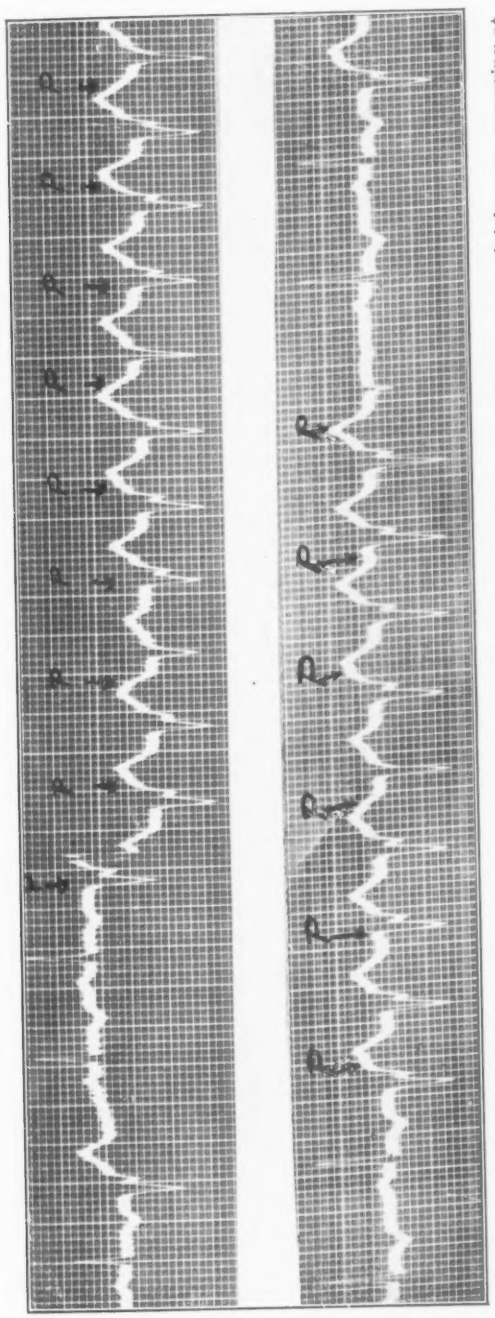

Fig. 1.—Two strips, both Lead II, show the auricular and ventricular dissociation. The P-waves are plainly seen, occurring at a slower rate than the ventricular impulses. Isolated extrastoles are also seen. In the second strip, the first beat of a paroxysm is coupled to a preceding normal beat by the same interval as is in isolated extrasystole.

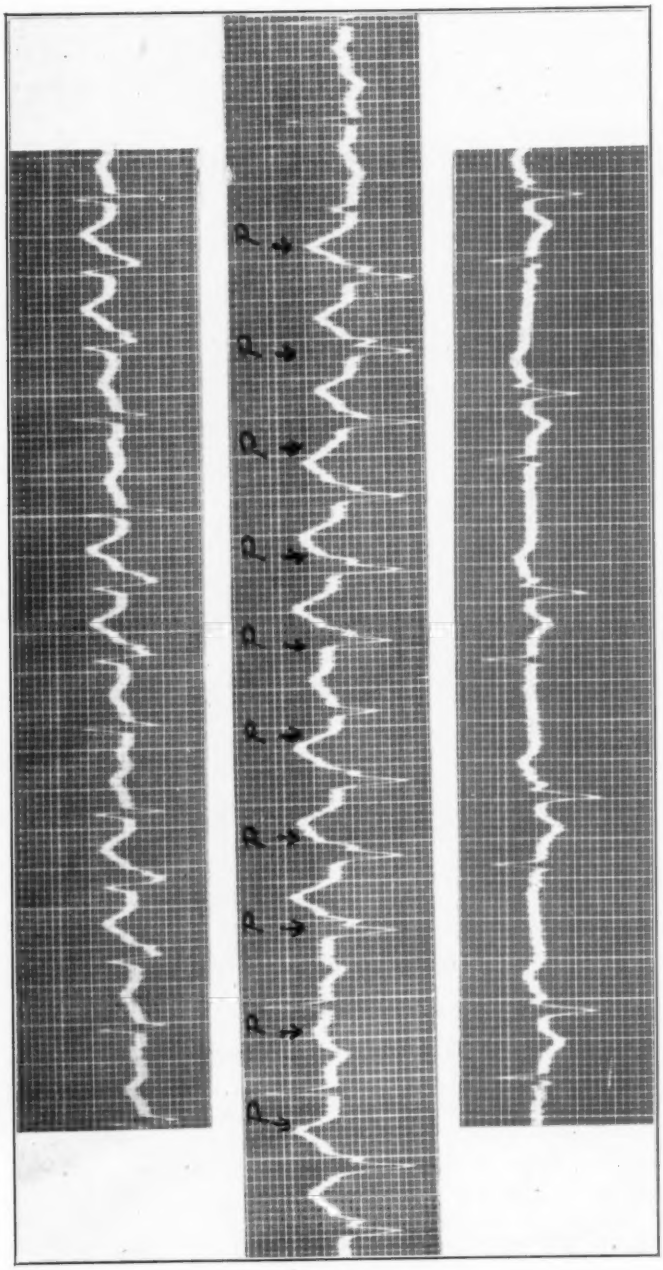

Fig. 2.—The three customary leads. One short paroxysm of tachycardia is shown. At no place in the tracing are more than two normal beats seen in sequence. In Lead III, a bigeminal rhythm is seen. This tracing was obtained after treatment had been instituted.

The urine, blood count and blood chemistry were normal, and the Wassermann test was negative.

Electrocardiograms.—In Figs. 1 and 2 (both obtained after one week of hospitalization) the tachycardia is seen to be composed of widened, aberrantly shaped ventricular complexes that are entirely independent of, and more rapid than, the auricular waves. Moreover, there are isolated, premature beats, almost certainly ventricular in origin, which are identical in shape with the complexes making up the paroxysm. The latter beats are sometimes accurately coupled to preceding normal beats, and frequently the first beat of the paroxysm is coupled to a preceding normal beat by the identical interval.

Fig. 3 is shown to illustrate the almost continuous nature of the paroxysmal disturbance as it existed before treatment was instituted, the tachycardia being

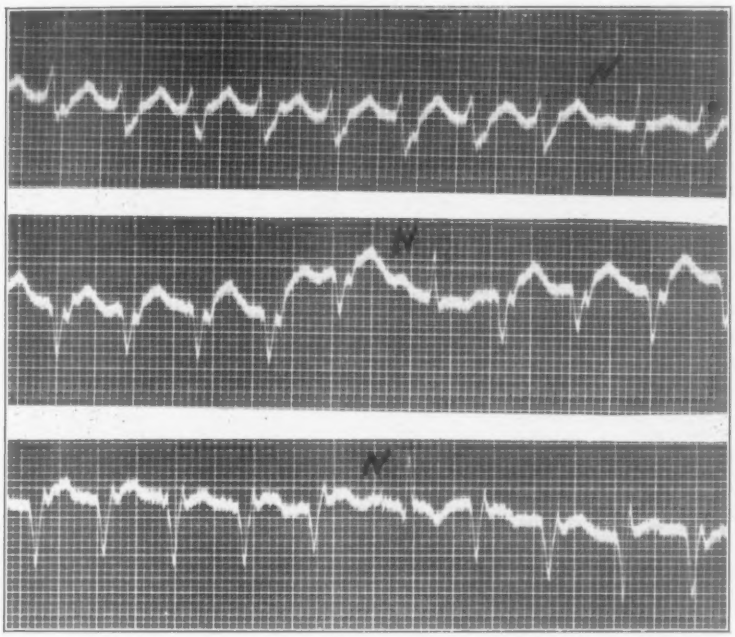

Fig. 3.—The three customary leads as they were observed before treatment. Tachycardia was present almost constantly. In each lead shown, only one normal supraventricular complex is seen. The T-waves of the normal complexes are inverted in Leads II and III.

interrupted only occasionally by single normal cycles. Until treatment was begun, the periods of tachycardia far exceeded the periods of normal rhythm.

Fig. 4 shows the electrocardiogram after the tachycardia had been abolished by quinidine.

The Effect of Drugs, Vagal Pressure and Exercise

Digitalis.—Four and one-third (4⅓) drams of the tincture of digitalis administered in a period of three days caused no change. Later, she was put on one dram of the tincture daily until signs of toxicity, in the form of nausea, headache and vomiting, appeared on the fifteenth day. On the fifth day of this régime and at various times afterward during the drug's administration normal rhythm appeared, but this was not firmly established; for such factors as exercise and emotional disturbance caused an immediate return of the tachycardia. More-

over, with the appearance of toxic digitalis effects, the tachycardia reappeared as the predominant mechanism. The most that can be said therefore is that digitalis in therapeutic amounts discouraged the tachycardia.

Quinidine.—The effectiveness of quinidine in controlling ventricular tachycardia has been well established.[18, 5, 7, 13, 8, 3] While our patient was having almost continuous paroxysmal tachycardia, the administration of quinidine sulphate was begun. After a total of thirty-five grains had been given in three days, a constant normal rhythm was restored with a ventricular rate of eighty-eight (88). The drug was then omitted and after three days paroxysms of tachycardia returned and persisted almost continuously during its omission. This procedure was repeated a number of times and always the arrhythmia disappeared on as much as five

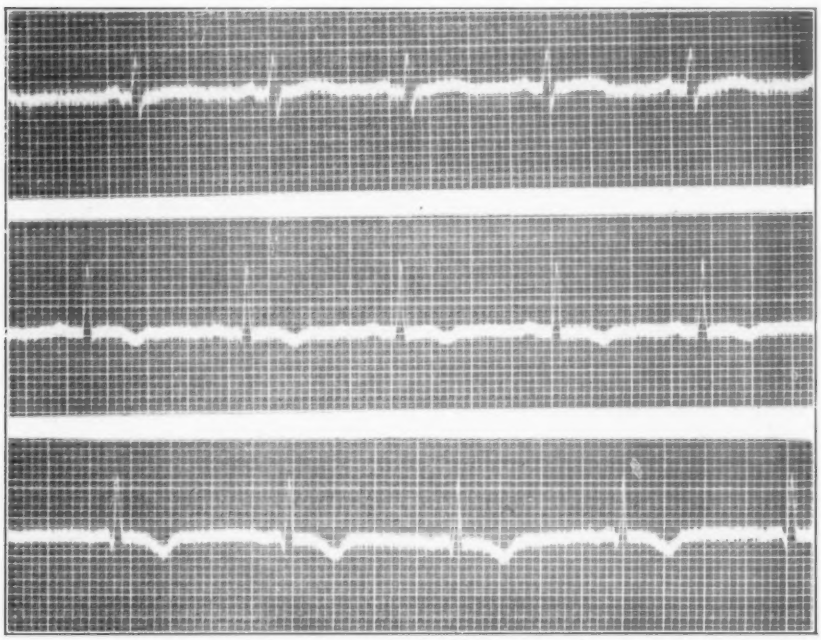

Fig. 4.—The three usual leads obtained after normal rhythm had been established under quinidine. The T-waves in Leads III and III are inverted.

grains of quinidine sulphate four times a day (frequently smaller amounts sufficed), and invariably returned when the drug was discontinued. The abolition of the arrhythmia under this drug was moreover apparently complete in that emotion, exercise, and atropine did not cause its reappearance. Under the circumstances of this case, we feared to give much quinidine for its possible untoward effect on the uterine muscle. These fears were justified inasmuch as signs of a threatened abortion did develop while the patient was receiving only nine grains of quinidine sulphate daily. After this episode, quinidine was not given again until after delivery.

Digitalis and Quinidine Combined.—A combination of 15 minims of the tincture of digitalis and 3 grains of quinidine sulphate three times daily restored and maintained a normal rhythm during its administration. In view of the response to each drug separately, it seems not improbable that the effects noted were really due to quinidine and not to the digitalis which was given with it.

Exercise, Vagal Pressure, and Atropine.—With the tachycardia almost continuous as it was before treatment was begun, we could see no effect of exercise. When the tachycardia had disappeared under digitalis, it promptly reappeared under the influence of exertion. While the tachycardia was absent under the influence of quinidine, we were unable to make it reappear by exercise or even atropine. Digital pressure over the vagus nerve in the neck had no demonstrable effect upon the ectopic rhythm.

We attempt to draw no conclusions from these observations. We had hoped to make more thorough observations after the puerperium was completed, but were unable to induce the patient to return for further studies after her discharge from the hospital.

Clinical Course

As the end of term approached, it was felt that cesarean section was the method of delivery that offered the fewest risks. This was accomplished successfully and uneventfully under spinal anesthesia by Dr. John McGlinn.

Fear of the possible depressing effects of quinidine on the respiratory center of the child led us to allow the patient to go into the operation with the tachycardia in full swing. Just before operation the tachycardia and periods of normal rhythm were intermingled as we had so often observed them. During the operation the tachycardia became entirely continuous with a rate of 150.

After delivery the abnormal rhythm continued to be present just as it had been during pregnancy and continued to be easily controllable by quinidine. Since her discharge we have been unable to make further observations, but have learned from a physician who has seen her from time to time that the tachycardia as shown by electrocardiograms is still present more than a year after our original observations were made. The last electrocardiogram that we ourselves made was obtained three months after delivery.

DISCUSSION

Incidence and Diagnosis of Ventricular Tachycardia.—This disturbance of the cardiac rhythm has always been regarded as rare. Its diagnostic features were somewhat confused until 1920, when Robinson and Herrmann[14] reported four cases and laid down certain criteria for a positive diagnosis. Submitting all the reported cases to examination, they accepted only eighteen as definite examples of ventricular tachycardia. Their criteria have been generally accepted, and since the publication of their paper there have been added a number of cases which complied with these. Recently Strauss[16] has reviewed all the reported cases and accepted sixty-five as being definite examples of ventricular paroxysmal tachycardia.

We doubt if the condition is as rare as the scarcity of reported cases would indicate. We have encountered thirteen instances among 16,000 electrocardiograms secured from 6,500 different patients, which fulfilled the criteria of Robinson and Herrmann.[14] It is probable that others have had a similar experience.

We have often wondered if A-V dissociation with a ventricular rate more rapid than the auricular might not under certain circumstances simulate a tachycardia of ventricular rather than of junctional origin. The circumstances under which this might occur would be those of an

accompanying widening of the ventricular complexes. There is evidence that junctional impulses may occasionally result in aberrant ventricular complexes,[4] the aberration at times even reaching the stage of widening.[20] If this did occur and the ventricular impulses were independent of, and more rapid than, auricular beats, ventricular tachycardia rather than the real condition might be diagnosed. However, when isolated extrasystoles are present and coupled to preceding normal complexes as they were in our case, a ventricular origin seems unquestionable.

Age of Onset.—While the number of reported cases of ventricular tachycardia is rapidly accumulating, few instances have been reported in young persons. Strauss[16] in his recent analysis found only three examples in persons less than twenty years of age. We have been able to find six reported examples among the reports of Wolferth and McMillan,[19] Jones and White,[5] Gilchrist,[3] Gallavardin and Veil,[2] and Anderson.[1] The youngest of any of these six cases was seventeen years of age. The age of our patient, sixteen years, is therefore, unusual.

Recently, Moore[11] has reported an interesting disturbance of rhythm in a boy of twelve years as an example of ventricular tachycardia. Study of the published tracings lead us to feel that this diagnosis on the evidence presented is somewhat open to question.

The Condition of the Heart in Ventricular Tachycardia.—In most of the reported cases there has been unquestionable evidence of cardiac disease. Levine and Fulton[8] state that ventricular tachycardia may occur on rare occasions in young people in whom there are found no signs of heart disease and in whom the only cardiac symptoms present are those associated with the tachycardia. They cite three examples: one reported by Scott,[15] one by Jones and White,[5] and one of their own.[8] However, Scott's patient was an invalid when she came under observation and, moreover, gave a history of a previous rheumatic infection. The case reported by Jones and White[5] showed definite cardiac enlargement. Levine and Fulton's patient[8] showed no demonstrable cardiac abnormality, but was forty-one years of age. In a report by Gallavardin and Veil,[2] the patient, a boy of seventeen, had no definite cardiac symptoms but the heart was enlarged. The example of ventricular paroxysmal tachycardia recently reported by Anderson[1] occurred in a boy of seventeen years, who showed no evidence whatever of heart disease. It is apparent, therefore, that ventricular tachycardia occurs but rarely in patients who present no demonstrable cardiac damage.

In our case we were unable to find anything by clinical study that could be interpreted as evidence of heart damage. X-ray examination showed the heart was definitely not enlarged, though by percussion it was on the borderline; but in one with the habitus of our patient and

with the addition of a seven months' pregnant uterus, we feel that the percussion measurements could be discounted even without the aid of the x-ray pictures. There is but one finding that leaves any doubt in our mind as to the absence of cardiac damage; this is the presence in the electrocardiograms of inverted T-waves in Leads II and III. We can only say of this finding that we know it can be the result of preceding cardiac damage; on the contrary, we see the change not infrequently in hearts that are considered to be unimpaired.

The Relation of Ventricular Tachycardia to Pregnancy.—It is not unusual to encounter extrasystoles during and apparently the result of pregnancy. Paroxysmal tachycardia on the other hand has been infrequently reported.[6, 10, 12, 17, 9] In those cases studied with the electrocardiograph, the origin of the tachycardia has been either auricular or nodal. The case of MacKenzie,[9] studied with the polygraph, was thought to be ventricular.

There is no evidence that establishes pregnancy as the cause of paroxysmal tachycardia of any variety. In most of the reported cases the disturbance had existed as a paroxysmal manifestation before pregnancy had begun. In our case the persistence of the tachycardia after the termination of pregnancy rules out gestation as the cause.

As a complication of labor, paroxysmal tachycardia, especially in diseased hearts, may be a serious matter as some of the reports referred to indicate. In patients whose histories indicate a serious circulatory response to attacks of paroxysmal tachycardia, it may be well at least to consider cesarean section as the method of delivery unless the disturbance is readily controllable. This means of terminating pregnancy was chosen in one of the cases of Meyer, Lackner and Schochet[10] and in our case. In both instances the results were highly satisfactory.

SUMMARY

A case of ventricular tachycardia is reported in which the following interesting features are to be noted: (1) the youth of the patient (sixteen years); (2) the absence of any definite evidence of a damaged heart; (3) the occurrence of the disturbance during pregnancy and its failure to disappear upon the termination of gestation; (4) the ready abolition of the tachycardia by quinidine; and (5) the successful and uneventful outcome of a cesarean section performed during the tachycardia.

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ANOMALOUS PAPILLARY MUSCLE ATTACHED TO PULMONARY VALVE OF HEART*

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IN 1896, Browicz reviewed the literature on anomalous chordae tendineae and papillary muscles occurring in the heart. He was the first to classify these anomalies and his classification is still the stand-

Fig. 1.—Gross appearance of the right ventricle of the heart. An anomalous papillary muscle is inserted into the anterior cusp of the pulmonary valve. The single chorda tendinea may be seen lying between the probe, which passes behind the anomalous papillary muscle and the base of the valvular cusps.

ard. The types are as follows: (1) normal papillary muscles; (2) so-called moderator band, stretching across the cavity of the right ventricle from the base of the anterior papillary muscle to the septum; (3) muscular chords running along the walls, usually of the ventricles, without any connection with valve curtains or trabeculae; (4) chords or

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TABLE I

REVIEW OF LITERATURE ON ANOMALOUS CHORDAE TENDINEAE AND PAPILLARY MUSCLES

YEAR	CLASS (BROWICZ)	REPORTER	CASES	AGE, SEX, YR.	SITUATION IN HEART	ORIGIN	INSERTION	CLINICAL DATA
1870	4	Mayne	1	Not given	Left ventricle, below aortic valve; 2 chordae	Not given	Not given	Loud apical systolic murmur
1876	4	Cuffer	1	35 F	Left ventricle	Anterior leaf of mitral valve	Wall of left ventricle below aorta	Basal murmur; mitral insufficiency
1878	4	Archer	1	Not given	Across aortic orifice	Not given	Not given	Not given
1879	4	Surbled	1	33 F	Left ventricle	Anterior papillary muscle	Behind mitral valve	Mitral insufficiency
1879	4	Demange	1	70 M	Right ventricle, anterior to tricuspid valve	Wall of right ventricle	Wall of right ventricle	Tricuspid insufficiency
1879	4	Veradini	1	Not given	Left ventricle, below aortic orifice to chorda	Wall of left ventricle	Wall of left ventricle	Loud systolic murmur
1882	5	Fowler	1	42 M	Left ventricle, left auricle	Left edge of foramen ovale	Left auricular appendage	Carcinoma of liver; heart negative
1884	4	Engel	1	Not given	Left ventricle, across aortic orifice	Wall of left ventricle	Aortic valve, posterior cusps	Aortic insufficiency, diastolic murmur
1892	4	Huchard	1	49 M	Left ventricle	Wall of left ventricle	Wall of left ventricle	Interstitial nephritis, basal systolic murmur, thrill
1896	5-(1) 4-(1)	Turner	2	Not given	One in left ventricle; one involving both valves	Near foramen ovale	Mitral valve	Not given
1896	5-(1)	Rolleston	3	1 M	Left ventricle	Left auricle	Left ventricle	Not given
	4-(2)			Not given	Left ventricle	Left papillary muscle	Right papillary muscle	Not given
				Not given	Left ventricle; 2 chordae	Papillary muscles of left ventricle	Mitral valve Left ventricle	Not given
				Not given				
1896	5	Griffith	1		Left auricle	Left side of foramen ovale	Left auricular appendage	Not given
1896	5-(2)	Bisiadecki and Feigl	2	Not given	Left ventricle	Near foramen ovale	Mitral valve	Not given

TABLE I—CONT'D

YEAR	CLASS (BROWICZ)	REPORTER	CASES	AGE, SEX, YR.	SITUATION IN HEART	ORIGIN	INSERTION	CLINICAL DATA
1897	3-(9)	Lewis	51	Not given	Thirty-two in left ventricle	Wall of left ventricle	Wall of left ventricle	Not given
	4-(35)			Not given	Twelve in right ventricle	Wall of right ventricle	Wall of right ventricle	Not given
	5-(7)			Not given	Seven in right and left ventricles	Wall of right ventricle	Wall of left ventricle	Not given
1901	5	de Vecchi	1	64 M	Left auricle	Edge of foramen ovale	Mitral valve, anterior cusp	Right lobar pneumonia
1902	4	Rössle	1	Not given	Left ventricle, multiple	Wall of left ventricle	Wall of left ventricle	Not given
1904	4-(2)	Poscharissky	2	Not given	Left ventricle	Papillary muscles of left ventricle	Aortic valve, posterior cusp	Not given
				M	Right ventricle	Wall of right ventricle	Pulmonary valve; right lateral cusp	Not given
1904	3	Sinivaldi	1	Not given	Left auricle, 3 chordae	Wall of left auricle	Left auricular wall	Not given
1926	5	Goforth	1	23 F	Right auricle; right ventricle; left ventricle	Right auricle	Aorta	To-and-fro-murmur
1928	4-(2)	Yater	2	Not given	One in left ventricle	Wall of left ventricle	Mitral valve	Not given
				Not given	One in right ventricle	Wall of right ventricle	Tricuspid valve	Not given
1928	4	Jäger	1	Not given	Left ventricle	Wall of right ventricle	Mitral valve	Not given
1931	4-(10)	White	12		Six in left auricle; four in right auricle	Not given	Not given	Not given
	5-(2)			Average 42	Both ventricles	Not given	Not given	Cardiac failure in five; pneumonia in two; other cardiac defects in nine

bands stretching across any one of the cardiac chambers from a papillary muscle or from one wall to the other, and inserting in a valve (and commonly giving rise to the high-pitched, musical, basal systolic murmur, transmitted upward toward the vessels of the neck and occasionally diastolic in time) and (5) anomalous chords extending from either auricle into the ventricle, originating from the edges of the valve over the foramen ovale and inserted into one of the auriculoventricular valves.

Table I contains a review of the literature on this subject from 1870 to 1931. Papillary muscles of the right ventricle attaching themselves to the pulmonary valve were reported in but 2.2 per cent of the cases found in the literature, and therefore constitute an uncommon cardiac anomaly.

REPORT OF CASE

The case to be reported is that of a man, aged sixty-nine years, whose weight was 215 pounds and whose height was 182 cm. The patient died of a disease totally unrelated to the cardiac anomaly, in life made no complaints relative to the heart, and on physical examination revealed no cardiac murmurs.

The heart weighed 400 gm. The foramen ovale was closed. Aside from the condition of the pulmonary valve, and sclerosis, graded 1, of the aortic valve and the anterior leaflet of the mitral valve, the valves and endocardium appeared normal. The right auricle was dilated, graded 2. There was sclerosis of the right coronary artery, graded 2, and of the left coronary artery, graded 3; the lumen of the latter was markedly stenosed. At a distance of 7 cm. from the apex of the right ventricle and 2 cm. mesially to the cut left lateral aspect of the right ventricle, there originated a thick, muscular band distinct from the myocardium (Fig. 1). It extended for a distance of 2.3 cm. and was inserted directly into the base of the anterior cusp of the pulmonary valve. The diameter of this band, at its origin in the wall of the right ventricle, measured 1 cm., whereas its diameter at the point of its insertion into the pulmonary valve was 0.5 cm. One chorda tendinea, measuring 0.8 cm., originated on the left side of this muscular bundle, and was inserted into the myocardium at the base of the line of juncture between the left lateral and anterior cusps of the pulmonary valve. This anomalous papillary muscle is of type 4, as described by Browicz. The presence of one chorda tendinea in this case clearly establishes this muscle bundle as being an anomalous papillary muscle and not an aberrant trabecula corneae. Its congenital origin seems to be clearly established.

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THE CLINICAL SIGNS OF OCCLUDING THROMBI OF THE LEFT AURICLE*

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IT WAS in 1890 that Von Ziemssen[1] suggested for the first time the possibility of recognizing the clinical manifestations of occluding auricular thrombi. He concluded from a study of three patients, that the presence of an obstructing ball or pedunculated thrombus in the left auricle could be diagnosed on the basis of circumscribed gangrene of the feet, cadaveric coldness and swelling of the lower extremities and absent or diminished arterial pulsations in the larger vessels of the legs. He attributed the latter signs to an extreme diminution of the blood flow from the left ventricle to the periphery by a thrombus superimposed on a pre-existing stenosed and narrowed mitral orifice. This pathological condition he found in all of his three cases. He based the development of gangrene of the feet on a similar mechanism although the possibility of marantic thrombi or emboli to the larger vessels of the legs could not be ruled out.

A few years later, Bozzolo[2] reported the case of a woman, forty-three years of age with mitral stenosis, in whom he established a diagnosis of an occluding thrombus of the left auricle and confirmed the findings at autopsy.

About one month after her recovery from a left hemiplegia, his patient began to complain of severe precordial pains and cough without expectoration. One week later, she experienced pain in her entire left leg and following that, the extremity began to swell and to feel cold and numb. Shortly after, the same signs appeared on the right side. A few days later, the left leg presented a mottled discoloration, livid in color, that extended from a little above the knee to the lower part of the leg.

When examined by Bozzolo, she showed marked cyanosis of the face and lips. Both feet were intensely cyanosed and had a greenish tinge which merged imperceptibly with a lighter area, pinkish in color, near the ankles. One of her legs presented a large necrotic area 3 cm. long and 1 cm. wide on its outer aspect near the lower part of the tibia. Pressure over the popliteal space of that leg was very painful.

The heart rate was fast and irregular. The radial arteries were barely palpable and only the right pulse was present.

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Autopsy revealed a large pedunculated thrombus of the left auricle in addition to the mitral stenosis. The vessels of the extremities were not opened.

In 1909, Battistini[3] diagnosed, during life, "thrombosis" of the left auricle in two patients with mitral stenosis. His first case was a male, forty-six years of age, who complained of coldness in his lower extremities ten days before his admission to the hospital. On examination, his legs showed large scars of a violaceous color without any definite borders. The superficial veins near these areas were distended. The femoral pulses could not be obtained, and the left radials and carotid pulses were not palpable. Unfortunately further details of this case are wanting.

At autopsy this patient revealed a large rounded thrombus of the auricle adherent to the parietal wall and obstructing a stenosed mitral orifice. There were also partial obliterating thrombi in the lumen of the femoral vessels and the popliteal artery.

His second case was a woman of forty-four years with mitral stenosis who shortly before her admission to the hospital complained of severe dyspnea and cyanotic discoloration of both her legs. On examination she showed gangrene of both feet. The right leg was cold and numb and devoid of all sensations. Her radial and carotid pulsations were weak and irregular. She died thirty days after the onset of these signs.

At autopsy, a large thrombus was found attached to the auricular wall by a long pedicle, which permitted it to play freely over the stenosed mitral orifice.

Another case, similar to these, although complicated by subacute bacterial endocarditis was seen by Lutembacher.[4] His patient was a woman twenty-eight years of age with mitral stenosis and a rapid and irregular heart rate who had lost the sight of her right eye. While under observation, she developed a gradual discoloration of her nose which at first was bluish in color and at the end of eight days became black and gangrenous. This dried, and was ready to drop off. Within two weeks several necrotic spots appeared on the face and forearms. Death took place three weeks after the onset of these signs.

At autopsy a ball thrombus was found present in the left auricle in addition to the stenosed mitral orifice.

In a man aged forty-one years, with mitral stenosis and auricular fibrillation, Aubertin and Rime[5] noted the appearance of purpuric spots on the lower extremities and palms of the hands followed within four days by a progressive increase in the discoloration of the last phalanges of the right hand. The oscillometric readings on both arms were only one-half of a division. Gradually the tip of the nose became discolored and cold. Death took place fourteen days after the appear-

ance of these symptoms, and at autopsy a large pedunculated thrombus was found over-riding a stenosed mitral orifice.

This diagnosis was suspected during life, emphasis being placed on the marked hypotension with disappearance of the first heart sound and the progressive asphyxia of the hands, feet and nose with cyanosis, coldness and the diffuse purpuric eruption. Unfortunately no examination was made of the vessels supplying the extremities.

In another patient with azotemia and progressive asphyxia of the extremities who died four days after the onset of these signs, these authors could not obtain a post-mortem examination to confirm their suspicions of an occluding auricular thrombus.

In a woman aged fifty-five years, with mitral stenosis and auricular fibrillation Covey, Crook and Rogers[6] noted the development of sudden pain and bluish discoloration of the entire left leg. Within a few hours, the pain became more severe, and it appeared to them that the presystolic murmur previously present had increased in intensity at the same time. A diagnosis of an occluding auricular thrombus of the ball-valve type was suspected and this was confirmed at autopsy. Permission, however, was not obtained for examination of the femoral artery of the left leg which they believed to have been obstructed by an embolus.

To this series we wish to add three more cases that have been seen by us during the past few years at the Montefiore and Morrisania Hospitals in all of which we suspected the presence of an occluding auricular thrombus from the nature of the physical signs present. Contrary to the beliefs of Welch[7] and Hewitt[8] we feel that such a diagnosis is possible although we would not draw a "clear" distinction between a ball and pedunculated thrombus. The recognition of these clinical signs is of importance for they presage death within a very short time after their appearance.

REPORT OF CASES

CASE 1.—F. F., a female, aged 44 years, was admitted to the Montefiore Hospital on Oct. 5, 1926, and died on Oct. 28, 1926. Her chief complaints were dyspnea, weakness, nausea and vomiting.

Previous Illness.—The patient became ill for the first time in May, 1917, when she entered the Presbyterian Hospital because of shortness of breath and weakness. She was readmitted to the same institution on four separate occasions within the next few years because of repeated attacks of shortness of breath, palpitation of the heart and swelling of the lower extremities. While there, on September 26, 1926, the tips of all of her toes and all of her fingers were noted to have become uniformly deeply cyanotic and very tender. This condition cleared up somewhat on the following day, but the first and second toes of the right foot remained deeply purple and gangrene set in.

Physical examination on admission to the Montefiore Hospital revealed a pale undernourished woman showing marked respiratory difficulty. Her neck veins were distended. The apical impulse of her heart was in the sixth intercostal space in

the left anterior axillary line. There was a marked apical systolic thrill. The first heart sound was almost completely replaced by a loud blowing systolic murmur. P_2 was accentuated. The heart rate was 140, the rhythm irregular and there was a pulse deficit of 28 beats per minute. The blood pressure was approximately 190 mm. of mercury systolic and 90 mm. diastolic. The edge of the liver was palpable in the right iliac fossa. The lower extremities revealed a moderate amount of edema, most of which was localized above the ankle.

The finger tips of both her hands were cyanotic, the purplish discoloration extending as far as the first joint. They all presented on the volar surface, darker, hemorrhagic spots which were extremely tender on pressure. Placing the hand in hot water did not change the color, although pressure on the pad of the distal phalanges caused blanching. The tips of all of her toes were similarly affected but not to as great an extent.

Both radial as well as the posterior tibial and dorsalis pedis arteries were visibly pulsating.

Course in Hospital.—On Oct. 8, 1926, two days after admission, she developed numerous red spots in the axillary region. These were irregular in outline, disappeared on pressure, but not with change in position. They increased in number following a thoracentesis of the right chest. Within the next two weeks, the signs of congestive heart failure became more prominent, so that one week prior to her death she showed extensive edema of both her arms and legs as well as marked ascites.

The tips of her toes and fingers turned black and became dry and puckered. There was progressive cyanosis and a darker discoloration of the tip of her nose, although no frank gangrene developed. At no time throughout the period of observation was there noted a fall in blood pressure. A pulse was felt in all of the large peripheral arteries until the day of her death.

An unusual observation was the failure of digitalis to slow the ventricular rate materially even though she received 3 c.c. of the tincture daily.

The patient died in coma, a little more than one month after the onset of these peripheral circulatory changes.

Autopsy Findings.—(Only the points of interest are reported here.) The heart was moderately enlarged and roughly globular. It weighed 700 grams. The epicardial surfaces all over were thin, smooth and glistening except for a slight opacity over both auricular appendages and a small milk patch at the upper posterior angle of the right ventricle.

The base of the heart was dilated, the right lateral border of the left auricle forming the upper border of the right side of the heart. The right auricle was markedly enlarged and the auricular appendage was firm. On section, the auricular cavity was dilated while the auricular wall was hypertrophied.

The left auricle was strikingly enlarged. Its capacity was judged to be approximately 300 c.c. It was partly filled with blood. After removing the unclotted blood, a large irregular thrombus, somewhat pyramidal in shape, was seen to extend from the tip of the auricular appendage as far as the orifice of the mitral valve. The thrombus was free except for its attachment to the auricular appendage and could be easily displaced from over the mitral orifice. It measured 10 cm. from tip to tip. It was of a firm consistency, its lower pole hugging the left leaflet of the mitral valve but extending almost across the orifice.

The right ventricular wall measured 5 to 7 mm. in thickness. There was marked dilatation with a slight hypertrophy of the conus of the pulmonary artery.

The pulmonary orifice measured 8 cm. and except for slight thickening of the corpora Arantii, there were no abnormalities.

The left ventricle was dilated and hypertrophied, its wall measuring from 10 to 15 mm. in thickness.

The mitral orifice was irregular in outline but admitted the tips of two fingers. The leaflets were all fused into a continuous and markedly thickened mass of tissue. There was a ridge of soft polypoid vegetations along the lines of closure. The chordae tendineae were all thickened, shortened and hyalinized. The papillary muscles were markedly hypertrophied.

The aortic orifice measured 6 cm. All three leaflets were moderately thickened and shortened, and in part calcified. The sinuses of Valsalva were slightly enlarged. Along the line of closure of all three leaflets, there were continuous ridges of warty vegetations similar to those of the mitral valves.

The coronary arteries were patent throughout.

The aorta and its tributaries showed no abnormalities and there were no thrombi or emboli in the large vessels of the extremities.

The vena cava and its branches contained no thrombi.

CASE 2.—F. S., a married woman, aged 42 years, was admitted to the Montefiore Hospital on Nov. 22, 1929, and died on Nov. 24, 1929. She was first seen in consultation with Dr. C. S. Mirkin in September of that year.

Previous Illness.—The patient had been perfectly well until two and a half years before (July, 1927) when she began to complain for the first time of recurrent attacks of palpitation of the heart and shortness of breath on exertion. She was treated with quinidine for several weeks and felt well enough to be up and about until Oct. 14, 1928, when she suffered a left hemiplegia. From this time on she showed auricular fibrillation, and it is stated by her physician that she responded promptly to rapid digitalization, her ventricular rate averaging 70 beats per minute three days later.

She remained in bed for three weeks and recovered almost completely save for numbness of the left hand which still persisted.

With the exception of several attacks of epistaxis, she remained free from symptoms until Sept. 4, 1929. When seen on that day, the patient was vomiting, she was disoriented, her speech was altered, her memory was poor, but she did not present any other neurological manifestations. She recovered from all of these symptoms within one week.

On the night of Nov. 4, 1929, the patient was awakened suddenly by a sharp lancinating pain in the midsternal region. When seen an hour later, she was sitting up in bed breathing with difficulty. The respirations were 34 and labored, the temperature was 101° F. and the heart rate was totally irregular and averaged 180 beats with a deficit of 40 beats per minute. There was a definite wheeze to her respirations. A slight but persistent cough was always accompanied by blood-tinged sputum. On the following morning, she brought up large clots of blood. From then on, the patient's dyspnea increased and her ventricular rate remained at over 100 beats per minute despite the fact that she received 54 c.c. of the tincture of digitalis within twenty days.

Throughout the rest of her period of observation, she complained of periodic seizures of pain over her precordium. These were constricting in type, radiated to the back and then to left side of the neck and ear. Occasionally, the pain would radiate to the inner side of the arm and remain there. More often, a constricting sensation of the left arm above the wrist would be her only symptom. She obtained great relief from small doses of morphine sulphate. With the persistent high ventricular rate which was refractory to large doses of digitalis, signs of congestive heart failure developed very rapidly. The liver became large and tender to pressure, there was swelling of her legs, and the only method of relieving her dyspnea was to have her sit up with her head bent far forward and reclining on her folded arms.

Throughout this time, her mentality was fairly clear. She answered questions rationally but her memory was very poor.

On Nov. 17, 1929, she was suddenly awakened with a severe pain in her right leg and a dull aching pain in her back. At 9 P.M. of that day, examination revealed nothing unusual in that limb except some lack of sensation to the pin-prick test. The right femoral artery was pulsating and there was a visible pulsation in the right dorsalis pedis artery.

On the evening of Nov. 21, 1929, she complained of a persistent severe headache followed by nausea, vomiting, incontinence of feces and of urine, and at one period of the day her eyes were seen to roll to one side, she foamed at the mouth and could not speak. At 4 A.M. of the following morning she was perfectly rational but complained of numbness in both her legs from the hips downward.

When seen at this time, physical examination revealed a slight pitting edema of both lower extremities. There was pulsation in both femoral arteries. The apical impulse of the heart was in the sixth intercostal space in the anterior axillary line. The heart sounds were of good quality, the first sound being partly replaced by a loud blowing systolic murmur. The blood pressure was approximately 190 mm. of mercury systolic and 110 mm. diastolic, and it was practically the same on both sides. There was moderate congestion at the bases of both lungs. The liver edge was palpable 8 cm. below the costal margin. The ventricular rate was 185 beats per minute with a pulse deficit of almost 50 beats.

She was seen again on Nov. 22, 1929, when she complained of a very severe pain in the whole of the right leg. At this time the entire limb had a cadaveric appearance with mottling and marmoration of the skin from the hips to the toes. No pulsation was felt in the femoral or the dorsalis pedis arteries. The limb was cold to touch and was covered with a clammy sweat. There were diminished muscular power and loss of sensation on this side.

When reexamined at the hospital, on the afternoon of that day, both legs appeared normal. Although several of our associates were inclined to believe that she had suffered an embolus to the right femoral artery in the morning, they could not explain these signs in the afternoon.

Twelve hours later, the patient was seen in shock. She was conscious but could not talk. Her face had an anxious expression and was of a ghastly color. There was marked distention of her neck veins. The heart rate was very rapid and irregular. The right pulse was not palpable. The left was barely felt. Both of her femoral arteries were now pulseless, and oscillometric readings were not obtainable at any level on either lower extremity.

The oscillometer readings on the upper extremities revealed only slight movements in the left arm but none in the right. The color of the lower half of the body had a post-mortem appearance, pale with mottling and the cyanosis that is usually seen in patients shortly after death. It was cold and clammy to touch. Five minutes after these examinations, complete positive oscillometer readings were obtained on all extremities and there was a return of all pulses to the touch. The color of the skin appeared normal again.

Throughout the rest of this day (Nov. 24, 1929) the patient presented on and off, such symptoms and signs as were described above and at one time, during the absence of all pulsations, short tonic and clonic convulsions were noted in various parts of the body.

The heart sounds, the heart rate and rhythm presented unusual clinical features during this time. When pulsations were not palpable in the peripheral vessels, there was almost complete absence of heart sounds. The rate was extremely rapid averaging 200 beats per minute, but at times on listening over the precordium the heart sounds could barely be heard.

The patient died in coma on the evening of the same day.

Autopsy Findings.—The heart was enlarged and weighed 410 grams. The epicardial surfaces all over were smooth and glistening. Both auricles and ventricles were moderately dilated.

In the left auricle, there was found a round, fairly smooth, red, spherical thrombus about 4.5 cm. in diameter. Its lower pole facing the mitral valve was slightly indented into a mould that fitted the orifice very snugly. On section, the thrombus presented a large area of soft spongy grey matter in its interior. There were no other thrombi in the auricles. The mural endocardium was smooth everywhere, and there was no evidence of any rough areas in the auricular appendage.

The mitral valve showed considerable thickening, the edges appearing rolled and the cusps were slightly fused. There was a row of extremely fine warty vegetations along the line of closure.

The left ventricle was moderately thick and its endocardium presented in several places small hard calcified areas varying in size from 1 to 3 mm. cross section.

The aortic cusps were markedly thickened by very large, irregular, partly calcified vegetations which projected from both surfaces of the cusps. There was marked fusion of the commissure of the posterior and anterior cusps which were particularly thick and rolled at the edges.

The left coronary artery formed only the anterior descending branch. The right coronary artery divided immediately into two shortly after its origin.

The right ventricle measured 5 to 7 mm. at the region of the conus.

The tricuspid valve was slightly thickened and distinctly rolled at the free edge. The chordae tendineae were slightly shortened and thickened.

There were no signs of any thrombi throughout the entire length of the aorta or its main large branches. The femoral arteries were patent throughout.

Case 3.—C. B., a male, aged 62 years, was admitted to the Morrisania Hospital on Dec. 31, 1930, and died on Jan. 5, 1931.

Previous Illness.—The patient had been well and working as a butcher until two months before when he began to notice progressive swelling of the lower extremities and shortness of breath on exertion. The swelling of the lower extremities subsided at night or when he was at rest. During the past month he had noticed ulcerations of both legs near the ankles, and these had increased in size and had not yielded to treatment although the patient had been in bed for the past month. Attacks of paroxysmal shortness of breath had set in during the past few weeks, and these had increased in frequency and had not been relieved with rest in bed.

Physical examination on admission to the Morrisania Hospital revealed an old man extremely short of breath with marked edema of the lower extremities. The skin over both legs was smooth, stretched and cracked in several places where edema fluid was escaping. The left leg presented on its outer aspect a little above the ankle a large ulcer about 4 cm. in diameter. It was covered by a black scab at its inferior margin. The right leg presented an ulcer about 2.5 cm. in diameter which extended only through the superficial layers of the skin.

His lips and ears were markedly cyanotic. There was active arterial pulsation on both sides of the neck. The apical impulse of the heart was in the sixth intercostal space in the anterior axillary line. The heart sounds were of fair quality, the first sound being replaced almost entirely by a loud blowing systolic murmur. The heart rhythm was totally irregular with an average ventricular rate of 145 beats per minute and a pulse deficit of 45 beats. The pulses were unequal and irregular.

The blood pressure was approximately 140 mm. of mercury systolic and 80 mm. diastolic and it was practically the same on both sides.

The abdomen was soft and the liver edge was palpable 6 cm. below the costal margin in the midclavicular line. There were no signs of shifting dullness in the flanks.

Course in Hospital.—On Jan. 2, 1931, the patient complained of severe and excruciating pain in the whole of the right lower limb from the thigh downward. He was irrational and at times uncontrollable. The pain disappeared on the evening of that day.

On the following morning both his legs assumed a deep bluish discoloration extending from the ankle upwards almost as far as the region of the lower pole of the patella. The femoral pulses, however, were both palpable but weak. His ventricular rate was 164 beats per minute and there was a pulse deficit of over 50 beats despite the fact that he had received large doses of digitalis in the previous two days.

At 8 A.M. of Jan. 5, 1931, the patient was unusually dyspneic and disoriented. He could hardly talk. His right hand was cyanotic and presented a uniform violaceous discoloration extending as far as the middle of the forearm. The radial pulse on this side was palpable, and at first it was thought that this discoloration might have been due to pressure since the patient favored the right side.

At 2 P.M. of this day, his respirations had increased and became labored. The patient was in a stuporous condition and could hardly be aroused. The cyanosis of his right arm noted in the morning had become deeper and now extended almost to the lower part of the forearm. The fingers were almost black. It was also observed that the left hand had now become very dark in color. Both of his hands were very cold and clammy. The pulses were still present, however, but barely palpable. There was absolutely no change in the intensity of his heart sounds. Unfortunately an oscillometer was not available at this time.

The legs had now assumed a uniform black discoloration as far up as the knees.

The patient died in coma at 7 P.M. of this day.

Autopsy Findings.—(Only points of interest are reported here.) The heart was markedly enlarged and it weighed 870 grams. The parietal pericardium was thickened and adherent to the visceral pericardium at the apex and at the base of the heart.

The left auricle was markedly enlarged. The left auricular appendage was firm and prominent. On opening the left auricle, a cylindrical thrombus 2.5 cm. in diameter was seen to extend from the tip of the auricular appendage as far as the orifice of the mitral valve. It measured 9.5 cm. from tip to tip. Its lower pole was conical in shape and fitted directly over the mitral orifice. In this region the thrombus was of a different color than the rest, and it was faceted with the imprint of the mitral valves on it. The thrombus could be easily displaced from over the orifice since it was attached only to the auricular appendage.

The mitral orifice admitted the tips of two fingers. The leaflets were smooth at their edges and no vegetations were present.

The right ventricle was hypertrophied and measured 8 mm. near the conus.

The left ventricle was concentrically hypertrophied and its wall measured 15 mm. in thickness near the apex.

The aortic orifice measured 6.5 cm. in circumference. The aortic leaflets showed many calcified nodules and the commissures of the valves were thickened.

The coronary arteries were slightly tortuous and thickened but patent throughout.

The aorta and its tributaries showed no abnormalities, and there were no thrombi or emboli in the large vessels of the extremities.

The vena cava and its branches contained no thrombi.

DISCUSSION

The peripheral circulatory disturbances following obstruction to the mitral orifice by an occluding thrombus of the left auricle may be of two types, either transitory or permanent. The transitory signs con-

sisting of temporary interference with the circulation to the extremities and cerebrum as observed particularly in our second case, are easily explained. When the lower pole of the loose left auricular thrombus became impacted into the mitral orifice, the diminished blood flow from the left ventricle caused by its obstruction as well as the rapid ventricular rate, resulted in absent pulsations of the peripheral vessels and the cadaveric appearance of the extremities supplied by them. It was at such times that the patient's sensorium was disoriented and mild convulsive seizures were noted. The first heart sound practically disappeared when this occurred.

It is difficult, however, to explain why sometimes only one of the lower limbs lost its circulation temporarily while at other times no pulsations or oscillometric readings were obtainable in any of the limbs. The total absence of gangrene in this patient was probably due to the fact that occlusion of the mitral orifice by the loose ball thrombus was not complete and was present for short periods at a time only, thus permitting the reestablishment of the peripheral circulation with adequate nutrition to the extremities.

Of particular interest in this case is the mode of death which was gradual and not sudden as is suspected in the presence of a loose thrombus of the left auricle. She died after she had been in coma for some time.

The more permanent circulatory disturbances observed in the other two patients came on rapidly. Their appearance undoubtedly coincided with a sufficient growth of the lower poles of the pedunculated auricular thrombi to encroach upon the mitral orifice. In this manner the nutrition of the tissues farthest away from the heart was gradually diminished and resulted in gangrene of the terminal phalanges of the fingers and toes in one case, and gangrene of the hands and feet in the other.

In the patient with hypertension (Case 3) this was probably more rapid than in the case with mitral stenosis (Case 1).

It should be mentioned here that another factor responsible for the appearance of gangrene in such patients may be the development of marantic thrombi in the larger vessels of the lower extremities as a result of the poor peripheral circulation caused by the interference with the blood flow to the left ventricle and consequently to the limbs. Such a condition is a rare association with congestive heart failure, but it was found in Battistini's first patient.[3] Obviously embolic phenomena to the extremities must be ruled out, but these are usually single and affect only one limb at a time.

Ulcerations of the legs, seen in our third case, were also noted by Bozzolo,[2] but in both instances it was probably part of the gangrenous destruction of the tissues due to the very poor circulation. We have never seen gangrene or ulcerations of the leg develop solely because

of edema of tissues. In our case the main large vessels of the legs supplying these regions presented no emboli or thrombi at autopsy.

The postulate that the underlying pathological lesion must necessarily be a mitral stenosis[1] in order that gangrene develop in the course of an occluding auricular thrombus is not tenable since one of our patients in whom these signs were present had hypertension during life and post-mortem examination did not reveal any stenosis of the mitral orifice.

There is one phenomenon which all of these cases including those reported in the literature present and that is a rapid and irregular heart action with a marked pulse deficit due in every instance to auricular fibrillation. This undoubtedly predisposes and favors the formation of auricular thrombi since such thrombi are not found so commonly in patients with normal sinus rhythm. The factors, however, responsible for the formation of either ball or pedunculated thrombi are still unknown.

In none of our three cases was it possible even with use of very large doses of digitalis to slow the ventricular rate materially.

The prognostic significance of occluding auricular thrombi is of great importance. The presence of severe peripheral disturbances associated with such thrombi usually presages death within a very short time after the onset of their appearance. None of these patients has been known to live longer than a little more than a month at the most.

SUMMARY

1. Three patients are reported in whom a clinical diagnosis of an occluding thrombus of the left auricle suspected during life was confirmed by post-mortem examination. Two of these patients had mitral stenosis and one had hypertension.

2. In one patient the diagnosis of a ball valve thrombus was suspected on the basis of the sudden and transitory appearance of peripheral vascular disturbances such as uniform discoloration with cadaveric appearance of the extremities at a time when pulsations of the vessels to these limbs were totally absent. During such periods the sensorium was disoriented, the patient was in mild shock, the skin was cold, moist and clammy, and the first heart sound was inaudible, while oscillometric readings of the limbs affected revealed no movements.

3. A diagnosis of an occluding auricular thrombus was based in one case on the rapid development of uniform discoloration with gangrene of the terminal phalanges of the ten toes and ten fingers which persisted for more than a month until death. In the other case this diagnosis was based on the rapid progressive appearance of pain and gangrene of the legs and arms with weak pulsations in the vessels supplying these limbs.

4. All three patients showed auricular fibrillation with a rapid ventricular rate and were refractory to large doses of digitalis. It is probable that the irregular heart action favored the rapid development and increase in size of the auricular thrombi. The circumstances responsible for the formation of ball or pedunculated thrombi are still unknown.

5. The mechanics of gangrene formation in the presence of an occluding thrombus of the left auricle is based upon its obstruction of the mitral orifice causing a diminished blood flow to the periphery. This is enhanced by the further incomplete diastolic filling of the left ventricle because of the rapid and irregular heart action.

6. The onset of such peripheral disturbances in patients with heart disease is of grave prognostic significance for death takes place shortly after their appearance.

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A CASE OF ACUTE CORONARY OCCLUSION WITH ROENTGENOGRAPHIC EVIDENCE OF THE EARLY DEVELOPMENT OF AN ANEURYSM OF THE LEFT VENTRICLE*

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THIS case of coronary occlusion is presented because of the paucity of symptoms as compared with the extent of the pathological changes present; the evidence of the presence of an aneurysm of the ventricle, coming on seemingly during the acute myomalacic stage; and the occurrence of the lesion in a relatively young individual.

CASE REPORT

M. S. Male; aged 35 years; was admitted to the general medical service of Dr. J. Rosenthal, on November 20, 1930, complaining of pain in the left chest anteriorly and a slight hacking cough for four days previously. He had enjoyed perfect health until two weeks prior to admission. At that time he had an attack of sticking, burning pain in the epigastrium which occurred about half an hour after a meal and which lasted only a few minutes. He complained of sour eructations and belching; no tarry stools, vomiting or jaundice were noticed. Following this the patient felt well and worked for nine days. Five days before admission, while in the subway, he was suddenly seized with an attack of nausea and vomiting and a sternal burning and pressure, which was not severe, but which was accompanied by a fear of impending death. He could not go on, an ambulance surgeon was called, and he was removed to the County Hospital. The vomiting disappeared immediately, and the nausea persisted for a whole day; the sternal pain, which was not severe, was relieved in a few hours by a hypodermic of morphine. The following day the symptoms almost all subsided and within forty-eight hours the patient left the hospital against advice. Because of a slight hacking cough and slight pain in the left chest anteriorly, the patient thought "he had caught a cold" in the hospital and sought the advice of a physician. Hospitalization was advised because the patient was running a temperature of 101° to 102° F.

Family History.—Negative. Married eight years; wife had two children, both living and well.

Past History.—The patient was a truckman and worked hard. He smoked about three cigars and drank about three cups of coffee each day. He recalled no serious illnesses nor any surgical procedures. No cardiac pain, palpitation, dyspnea on exertion or edema prior to the onset of the present illness.

Physical Examination at the time of admission revealed a muscular young adult male in good nutrition, of the sthenic type. He did not appear very ill. No dyspnea or cyanosis was noted. Pressure over the styloid processes elicited no response, indicating marked hyposensitivity. Libman[1] has emphasized the importance of estimating a patient's reaction to pain in the interpretation of his symptoms. This case illustrates the point well. The head showed no gross abnormalities. Eyes and ears were negative. The pupils were regular, equal and

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reacted both to light and in accommodation. The eye grounds were normal and showed no evidence of vascular sclerosis. Nose negative. The tongue had a thin whitish coat. Fauces and pharynx negative. Neck negative. The chest was slightly pigeon breasted. The lung fields were clear throughout. The heart impulse was visible and palpable somewhat to the left of the midclavicular line in the fifth space. The signs were more marked in this localized area both on auscultation and on palpation over the rest of the precordium. The borders percussed as follows: *left*—3 cm. from midsternal line in the second space, 8.5 cm. in the third, 11 cm. in the fourth and 12 cm. in the fifth; *right*—2.25 cm. from midsternal line in the second space and 3 cm. in the third and fourth. The heart action was regular, with a rate of 70. Heart sounds were somewhat distant and not of good quality; the first sound at the apex was barely audible; A-2 was greater than P-2; no murmurs were heard. The abdomen and extremities appeared normal. No edema was present. None of the stigmata of syphilis could be found. Right carotid sinus pressure caused a cardiac standstill for a short period.

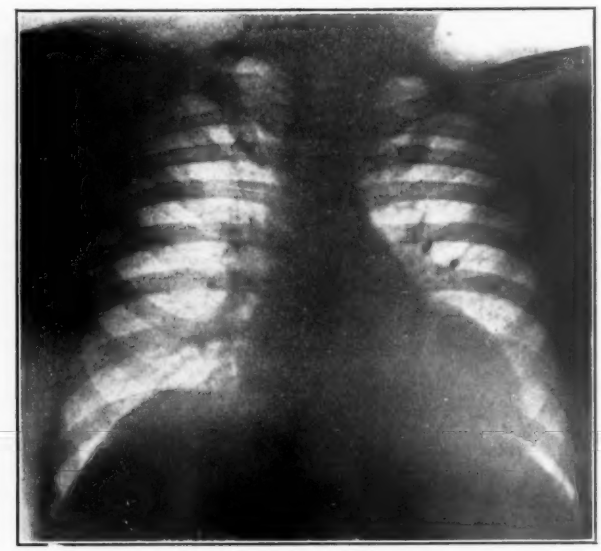

Fig. 1.—X-ray picture taken on the sixth day of illness, showing aneurysm of the left ventricle.

Laboratory Data.—The temperature ranged up to 101.4° F. the first six days of his hospital stay; from then on it was normal. The pulse rate ranged from 88 to 68. Blood pressure on admission was 110/70 mm.; on the eleventh day of his illness it was 90/60; thereafter, it gradually rose until on the day of discharge from the hospital his blood pressure was 125/80; two months later it was 170/110. On the eighth day of his illness his blood count was 17,800 white cells and 82 per cent polys.; on the twelfth day of his illness, or the eighth day of his stay in the hospital, his blood count was 8,200 white cells and 64 per cent polys.; from that time on the blood counts were normal. Sedimentation time was fifteen minutes eight days following the occlusion, and persisted low up to the eighteenth day of his illness when it was found to be sixty minutes; from then on it gradually increased. Red blood cell count was 5,120,000 and hemoglobin was 90 per cent. Blood Wassermann, Kahn, sputum, urine and blood chemistry determinations were all negative.

Roentgenological studies of the chest were made on the second day in the hospital and were reported as follows by Dr. M. G. Wasch: "The heart is considerably enlarged, especially to the left, where the apex assumes a rather globular contour and bulges in an unusual manner. Pulsation therein is synchronous with the cardiac borders so that the condition must be considered intrinsic. The configuration strongly suggests an aneurysm of the left ventricle." Frequent restudies were made over a period of three months and showed practically no change. The cardiac silhouette is presented in Fig. 1.

In Fig. 2 are given electrocardiographic tracings taken on the patient's third day in the hospital. They show a regular sinus rhythm with a rate of about 70. The P-wave is inverted in Lead III. The P-R interval is within normal limits. The main deflections in all leads are definitely notched. The deep S-1 and high R-3 indicate a right axis deviation. The S-T interval is elevated above the

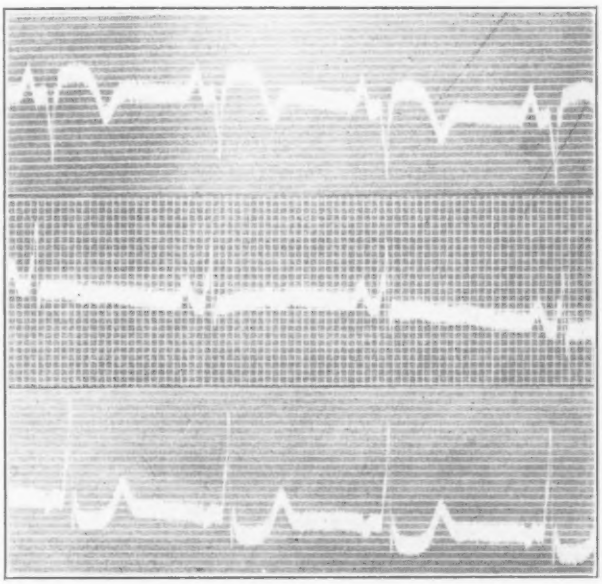

Fig. 2.—Electrocardiogram taken seven days following the coronary occlusion.

iso-electric line in Lead I and depressed below it in Lead III. The T-wave is large, coved and sharply inverted in Lead I and it is isoelectric in Lead II.

Electrocardiographic studies were made every other day up to the time of the patient's discharge from the hospital on December 16, 1930. The tracing taken on the day of discharge is presented in Fig. 3. Compared with Fig. 2 it shows a return of the S-T interval toward the base line and elongation with sharper coving of the T-1 which has become higher than the R-wave. T-2 is upright; T-3 is sharply pointed and higher than the R-3.

The few symptoms that the patient presented on admission cleared within the first seventy-two hours in the hospital. He showed no signs of cardiac embarrassment at any time. Activity was forbidden, but at the time of his admission to the hospital he experienced no difficulty whatsoever in standing or walking. His clinical course in the hospital was uneventful, and when last seen, three months following the onset of symptoms, the patient was enjoying moderate activity without pain or dyspnea. An electrocardiogram (Fig. 4) taken on February 21, 1931,

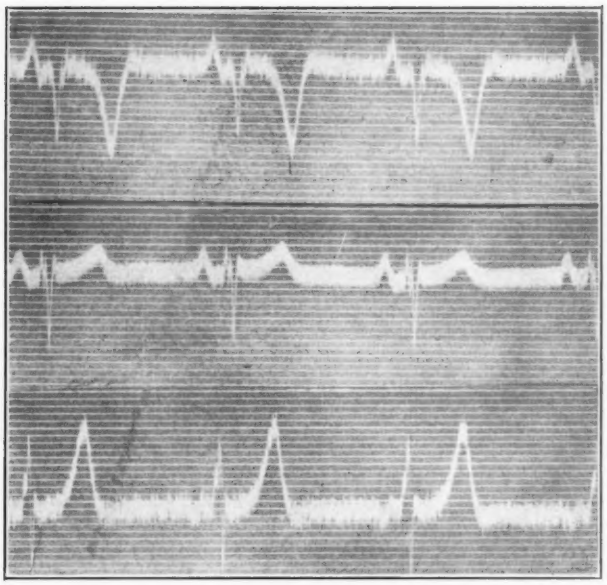

Fig. 3.—Electrocardiogram taken immediately before discharge from the hospital. Note the voltage of the T-waves in the first and third leads.

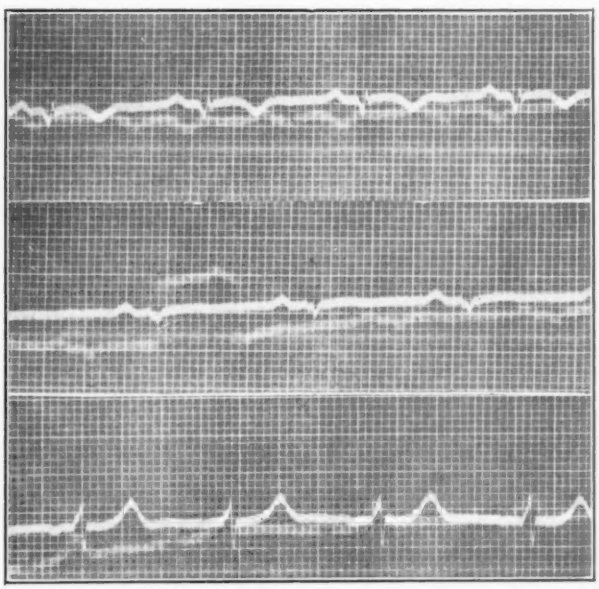

Fig. 4.—Electrocardiogram taken on February 21, 1931, at the Out-Patient Department. Note the extreme low voltage of the main deflections compared with the electrocardiograms taken while patient was in the hospital.

in the Cardiac Clinic, shows a regular sinus rhythm, ventricular rate 78. All main deflections are of very low voltage and slurred. T-1 is inverted; T-2 is iso-electric. These findings are indicative of rather severe myocardial disease.

COMMENT

The diagnosis of acute coronary occlusion and aneurysm of the left ventricle is, it appears to us, warranted by the evidence described above. Whether or not his recent illness represented the patient's first major cardiac insult may be questionable. To go with the marked x-ray and electrocardiographic changes presented, one would expect an extensive cardiac history. The patient was questioned very carefully in regard to the presence of cardiac pain, palpitation, dyspnea on exertion or edema prior to the onset of this illness, and he specifically denied having had any of these complaints at any time previously.

An aneurysm of the ventricle usually results from a dilatation of a healed fibrous area following a myomalacia cordis, and a definite period of time usually elapses between the original insult and the aneurysm. More often it is the result of several attacks. In this case, however, it would appear, from the history and the findings, that the aneurysm of the ventricle followed very shortly after this patient's cardiac insult. The complete absence of a history of previous attacks, as shown by the absence of pain, symptoms of cardiac insufficiency, and any of the pain equivalents of a coronary occlusion often found in hyposensitive individuals,[1] makes us feel that this lesion is the direct result of the recent coronary occlusion and is an aneurysmal dilatation of a fresh myomalacia.

In this diagnosis we have relied to a considerable extent upon the x-ray and electrocardiographic findings. The diagnosis of aneurysm of the heart is seldom made with certainty during life, but in this instance it appears to lack only post-mortem confirmation. B. S. Oppenheimer and H. Mann[2] state that the most common site for cardiac aneurysm is the apex of the heart and the middle or lower third of the anterior wall of the left ventricle, involving also the neighboring ventricular septum; with the next most common site as the posterior wall near the auriculoventricular ring. The aneurysm in this case is probably in the favorite site.

Another interesting feature of this case is the relative youth of the patient. Coronary thrombosis is rightly regarded as a disease of advanced years. One should bear in mind, however, the fact that it is not infrequently found in an individual under forty. Levine[3] gives the average age of 145 cases as 57.8 years, and presents in his series three cases (2 per cent) under forty years—two at thirty-nine years and one at thirty-six years. The patient observed by us was thirty-five years old. Lues was carefully excluded in this patient.

SUMMARY

An aneurysm of the left ventricle, the clinical and roentgenological symptoms of which were noticed in the first few days following a coronary occlusion in a comparatively young man, is reported.

We wish to thank Dr. Joseph Rosenthal for his permission to report this case.

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Department of Clinical Reports

THE ELECTROCARDIOGRAPHIC FINDINGS FOLLOWING LIGATION OF DESCENDING BRANCH OF THE LEFT CORONARY ARTERY IN MAN

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CONCLUSIONS as to the electrocardiographic findings in coronary artery disease have been arrived at by two methods: first by a study of electrocardiograms from individuals presenting symptoms of coronary disease and from those proved at autopsy to have coronary disease. This is what we might call a clinico-pathological study. A second method of approach has been study on experimental animals. The nature of the problem has made experimental study on man impossible. This paper is a report of findings under experimental conditions made necessary by an emergency. It presents a series of electrocardiograms obtained after ligation of the descending branch of the left coronary artery.

CASE REPORT*

J. H., a colored male aged 18 years, was admitted to the Emory University division of Grady Hospital on January 8, 1931. His chief complaint was stab wound of the left chest. At 10 P.M. on the date of admission he was stabbed in the left upper chest with a knife. He ran 30 or 40 yards and fell to the ground. He was picked up and brought to the hospital at once. At that time he was conscious but extremely restless. His clothing was covered with blood. Both pupils were dilated. His extremities were cold and clammy and radial pulse was imperceptible. His heart rate which at first was 90 per minute soon dropped to 60. He was thought to be in a state of cardiac tamponade and was sent at once to the operating room. Operation, by D. C. Elkin, was begun at 10:50 P.M. The heart was exposed and a penetrating wound of the pericardium found. With each cardiac systole blood spurted from this wound. The wound in the pericardium was enlarged and the pericardial cavity found to contain about 250 c.c. of clotted blood. There was a stab wound which penetrated the right ventricle near the interventricular septum. It ran parallel and very close to the descending branch of the left coronary artery in its middle third. In the course of the operation this artery was found to have been cut. Whether this injury had occurred at the time of the stab wound or accidentally during the operation could not be stated. The artery was tied by a double ligature. Operation was completed at 11:40 P.M.

*A detailed report of this case from the surgical standpoint is being prepared by Dr. D. C. Elkin for publication elsewhere.

Postoperative Course.—On the third day there was a definite to-and-fro pericardial friction rub which persisted until the sixth day. The operative wound healed nicely and all sutures were removed on the sixth day. At no time did the heart show any abnormalities of rhythm. On the eighth day he complained of pain in the epigastrium and examination showed a pleural friction rub in the lower left axilla and a few moist râles at the left base. These findings gradually decreased; his temperature reached normal, and on the twentieth day he was out of bed. He was dismissed from the hospital on February 6, 1931, and at present (March 26, 1931) is symptom free and apparently completely recovered.

Electrocardiograms were taken by the three standard leads just before operation (10:50 P.M.), just after operation (11:50 P.M.), and at frequent intervals during his stay in the hospital. Later tracings were obtained with difficulty because the

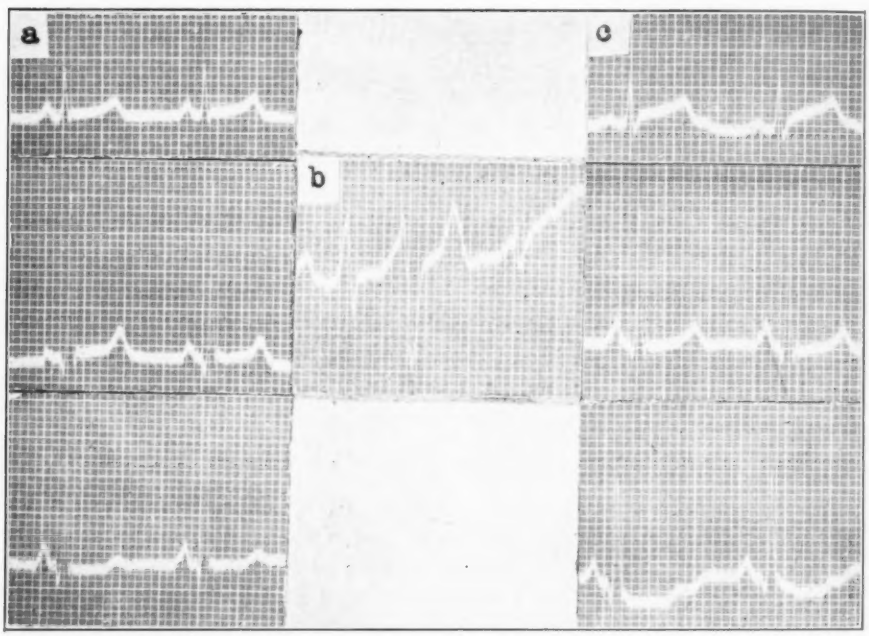


Fig. 1.—(Jan. 8) *a*. Before operation. Changing form of P in Lead II. *b*. During operation. Bizarre complexes. Ventricular premature beats. *c* Ten minutes after operation. Elevation of R-T in Lead I and depression in Lead III.

patient had to serve a period of one month in jail. An incomplete record in Lead II was taken during operation.

Before operation, during the state of cardiac tamponade, the only outstanding abnormality was a changing form of P-wave in Lead II (Fig. 1*a*). This was probably the result of irritation of the auricles by blood within the pericardium. During operation (Fig. 1*b*) the complexes were of bizarre forms and quite irregular. The predominating type of complex was a ventricular premature contraction. These were thought to be due to mechanical stimulation produced by the operative procedure. As early as ten minutes after the operation (Fig. 1*c*) definite change was observed in the R-T interval. In Lead I there was a slight definite elevation of the take-off of T while in Lead III the R-T interval was depressed and convex downward. On the second day after operation (Fig. 2) the T-wave showed maximum change. In Lead I and II there was a very high take-off. The

first definite change in Q-3 was noted at this time. It measured 3.4 mm. which was a definite increase over previous records. In subsequent records (Figs. 3 and 4) the T-wave was observed to gradually approach the iso-electric line. It became isoelec-

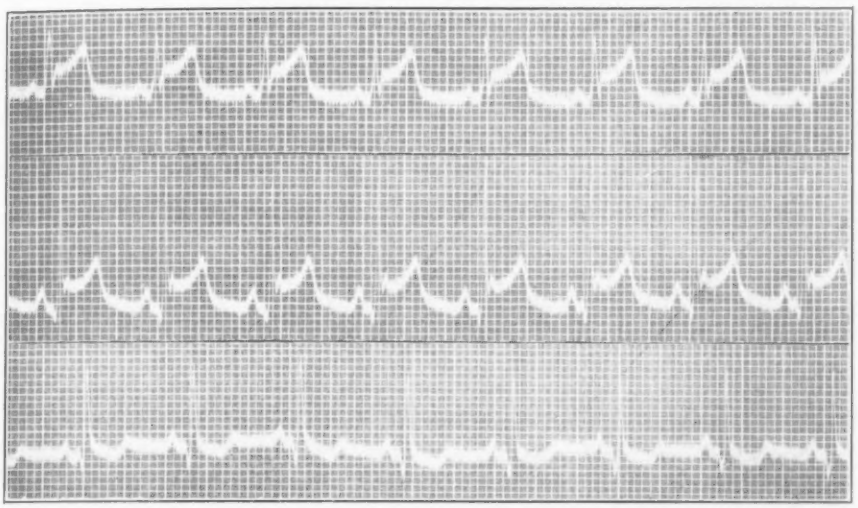
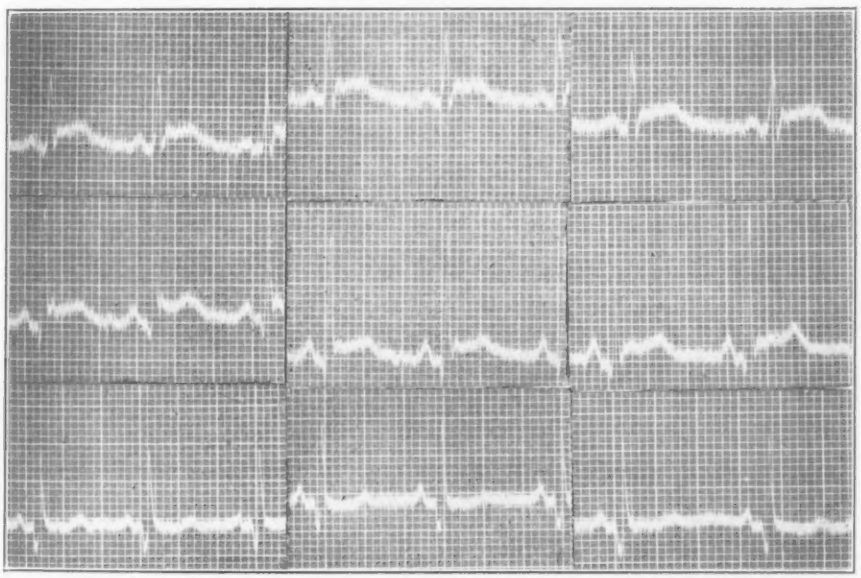


Fig. 2.—Second day (Jan. 10). Maximum change in R-T interval. Increase in Q-3.



Jan. 12. Jan. 13 Jan. 14

Fig. 3.—Fourth, fifth and sixth days. T gradually approaches iso-electric line. Q-3 shows maximum change on fifth day.

tric on the twelfth day and inverted sharply on the sixteenth day. On the seventy-seventh day it had become upright in all leads. The Q-wave in Lead III showed a progressive relative increase up to the fifth day. From the fourth to sixth days inclusive it was as much as 25 per cent of the greatest deflection of R (Lead II). From

that time on it gradually decreased. These changes may be followed in Fig. 5. The values represented in the curve are averages from measurements in seven complexes

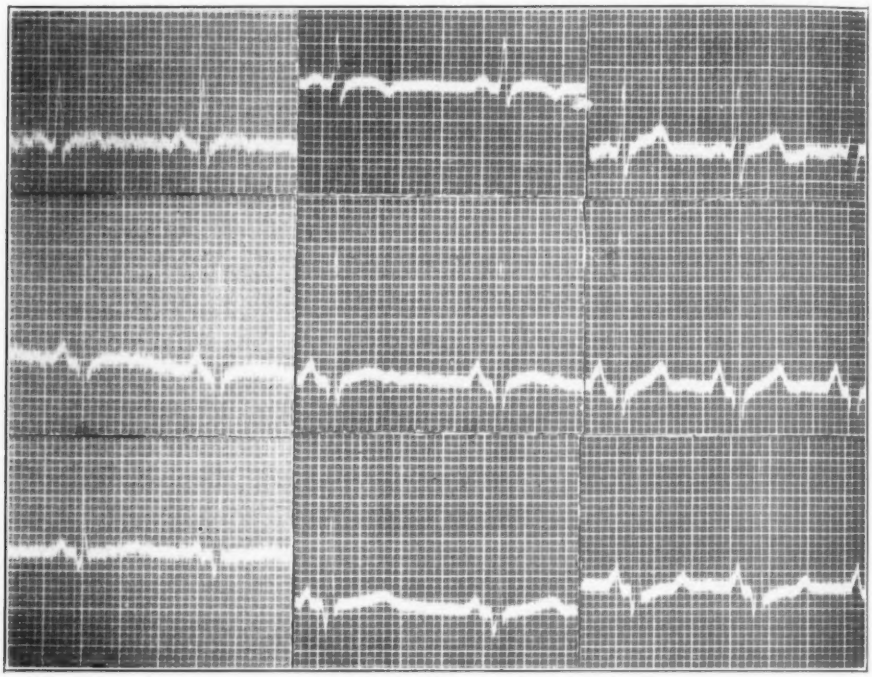


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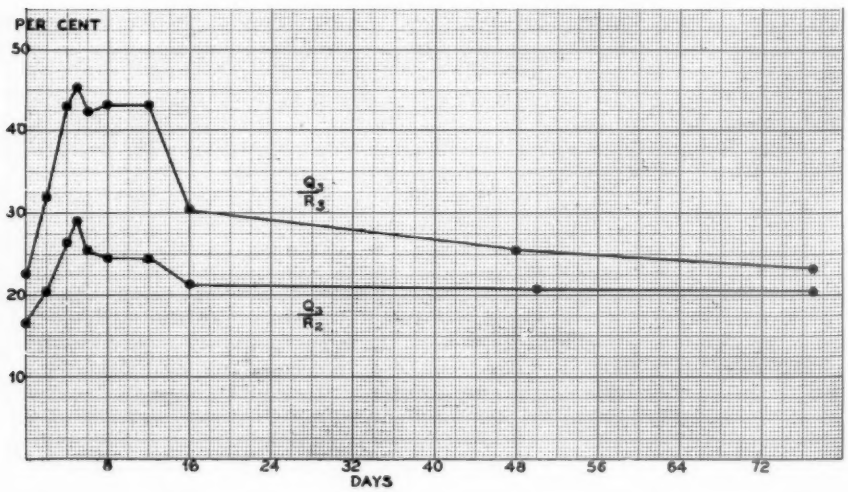
Fig. 4.—Twelfth, sixteenth, and seventy-seventh days. Iso-electric T changes to sharply inverted T and then becomes upright again.



of each lead. With the progressive increase in magnitude of Q-3 there was also a slurring of this same wave at its apex. With decrease in magnitude the slurring decreased.

COMMENT

A report of one case similar to the above was made by Smith[1] and Davenport.[2] Their first electrocardiogram was obtained on the nineteenth day after operation. At that time there was inversion of T in all leads. The T-wave was found upright after eight and one-half months.

Pardee[3] has recently called attention to a relative increase in size of the Q-wave in Lead III in records with a left axis deviation or a normal axis. He found that the majority of such records were obtained in patients with the anginal syndrome. Along with the increase in Q-3 in such patients he often found inverted T in Lead III or in Leads II and III. Where Q-3 is greater than 25 per cent of the greatest R or S it is considered abnormal. Wilson[4] has referred to a large Q in Lead III occurring in coronary occlusion.

The curve shown in Fig. 5 is not presented with the idea in mind that its form, duration or magnitude of changes represents what might occur in a similar case or in a patient with coronary occlusion, but it does definitely illustrate that the Q-wave in Lead III has undergone considerable change. The changes in the R-T segment do not differ from those often described before. Decrease in amplitude of QRS was not a remarkable feature of this case. Abnormalities of rhythm were not observed.

SUMMARY

After the unavoidable ligation of the descending branch of the left coronary artery in man the following changes were observed in the electrocardiogram:

1. Changes in the T-wave. (a) Definite elevation of take-off in Lead I and depression of the R-T interval in Lead III within ten minutes after operation (probably thirty minutes after ligation). (b) Maximum change within forty-eight hours. (c) In Leads I and II it gradually descended on R and became isoelectric in twelve days, inverted in sixteen days, and upright in seventy-seven days.

2. Changes in the Q-wave in Lead III. (a) Increase in size. It exceeded 25 per cent of the greatest R from the fourth to sixth days. Maximum increase was present on the fifth day. (b) Slight slurring at its apex.

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PERSISTENT TRUNCUS ARTERIOSUS: CARDIAC HYPERTROPHY, DYSPHAGIA, DEATH ON ELEVENTH DAY

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CONGENITAL cardiac defects similar to the one here recorded have been previously described several times, yet the condition is of sufficient rarity to justify placing on record another instance. Dr. Maude E. Abbott[1] in 1927 had collected but 23 cases. Most persons with persistent truncus arteriosus usually die shortly after birth. The defect, however, is not always incompatible with the attainment of adult life and activity as instanced by the last two cases which we have seen reported. In one[2] the patient was twenty-five years of age when killed in an automobile accident, and the malformation was discovered only at necropsy. In the other[3] the patient died of a superimposed endocarditis at twenty-two years of age. The present instance is unusual in that dysphagia was the main symptom calling attention to serious disease. Roentgen-ray pictures were obtained, and from them some gross malformation of the heart was diagnosed during the brief life of the patient, although the exact nature of the lesion was not revealed until necropsy.

CASE REPORT

Clinical Findings.—P. J. E., white female. Delivery normal, weight 7 lb. No abnormalities noted at birth except a thick mat of black hair on head and small, poorly developed ears. Older children in family normal, living and well.

Cry at all times rather weak. Slight cyanosis present at all times with severe exacerbations of marked cyanosis several times a day. Never able to nurse well. Chief symptom, however, was an apparent difficulty in swallowing. The breast was taken rather eagerly at first but was soon released and was followed by an almost immediate regurgitation of milk through the nose and mouth, so that the child received but little nourishment during its eleven days of life. Between feedings there was an almost constant drooling of saliva, which at times accumulated in the throat to such a degree that a suction apparatus had to be used to prevent asphyxia. The symptoms were so suggestive of an obstruction of the esophagus that a catheter was passed, barium sulphate given and roentgen-ray pictures were made. (See Figs. 1 and 2. *E* points to barium shadow in esophagus, *S* to stomach.)

At eleven days of age the cyanotic periods increased in frequency and severity, and the baby grew weaker and died.

Roentgen-Ray Examination.—Antero-posterior view shows an enormously hypertrophied right ventricle and right auricle. The right auricle expands upward and to the left. The angle which normally exists between the ascending aorta and the right auricle is not seen as the auricle extends well to the right and upward. There is an immense expansion of the heart in the horizontal diameter. There are no recognizable subdivisions of the heart into its different parts. Lateral view shows the right ventricle enormously enlarged upward and forward, filling in the retro-

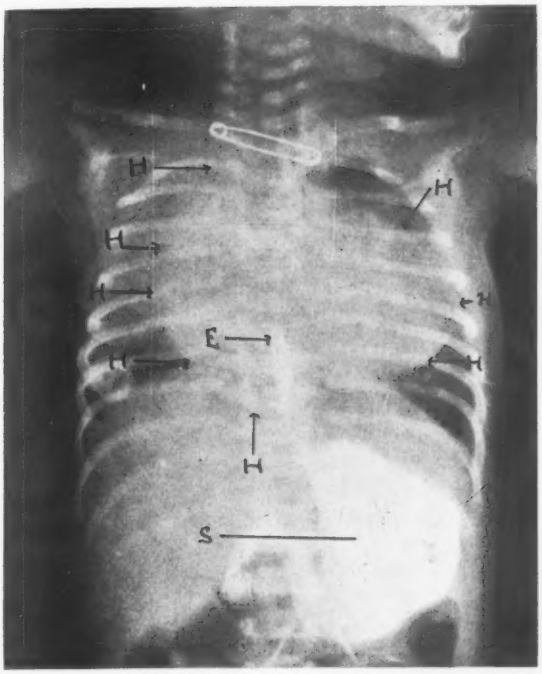

Fig. 1.—Antero-posterior roentgenogram of chest. Enlarged heart shadow, *H*; esophagus with small quantity of barium sulphate, *E*; stomach outlined by barium, *S*.

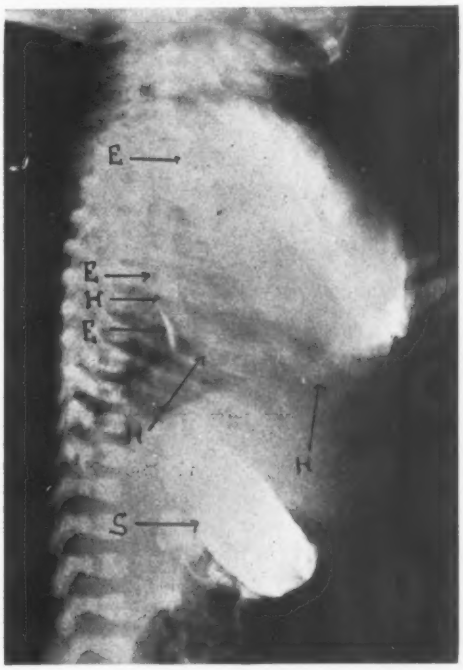

Fig. 2.—Lateral roentgenogram of chest. Enlarged heart shadow, *H*; esophagus, *E*; and stomach, *S*.

sternal space, which normally exists between the sternum and right ventricle. The retrocardiac space is almost filled by the large right auricle, and the esophagus (outlined by barium) is seen pressed upon (*H*, Figs. 1 and 2).

Post-Mortem Examination.—Body is that of a white female infant in the first few days of life. The scalp is covered with a thick suit of nearly black hair. The ears are small and placed rather obliquely on the head, the upper border of the pinna being somewhat diverted backward. The thorax is somewhat enlarged. On opening the thorax the heart is found to be enormously enlarged, occupying most of the anterior portion of the thoracic cavity, the lungs being displaced laterally and backward. There is a slight excess of dark colored serous fluid in each pleural cavity and a few small hemorrhagic spots are seen on each lung. The usual lobes of the lungs are indicated by rather shallow divisions. The cardiac notch of the

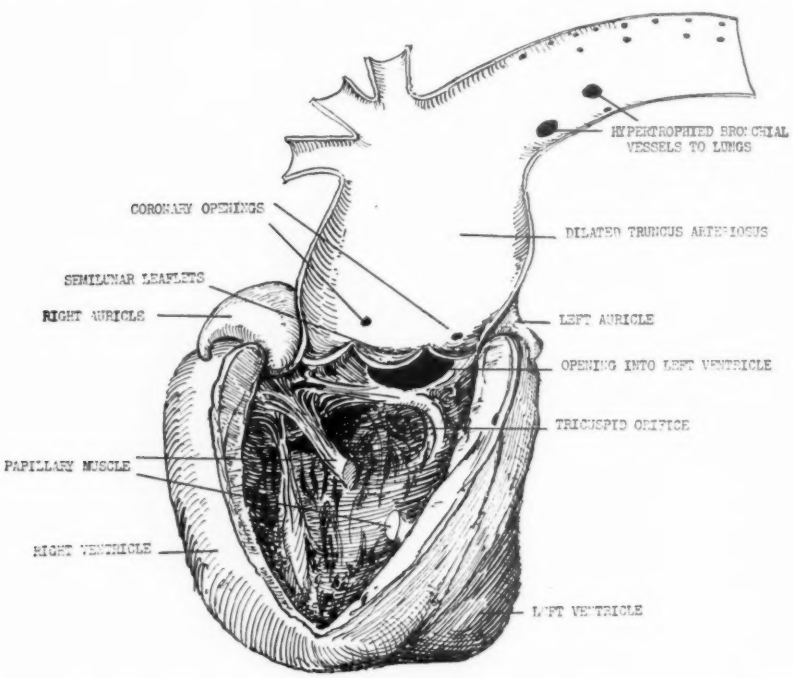

Fig. 3.—Anterior view of heart with the right ventricle opened and the truncus arteriosus opened throughout its extent.

left lung is very large, being nearly an equilateral triangle, the sides measuring about 35 mm. The middle lobe of the right lung is poorly defined and largely blended with the upper lobe. The lungs appear well aerated.

The pericardium is normal.

The thymus gland is normal in appearance and not enlarged.

The heart is greatly hypertrophied and the right side is noticeably larger than the left. The most conspicuous feature of the heart is the absence of the pulmonary artery and the origin of the truncus arteriosus from the right ventricle in the trihedral angle formed by the right auricle, the right ventricle and the left auricle. No vessel emanates from the left ventricle. In about the location where the aorta normally should arise from the left ventricle there is a well defined opening, admitting a lead pencil, diameter 8 mm., into the right ventricle and directly beneath the right posterior cusp of the aortic valve.

The right ventricle measures vertically 50 mm.; laterally, at the widest point, 35 mm.; wall at thickest point toward the base, 7 mm.; and at thinnest point toward the apex, 2 mm. The interior of its chamber aside from its large size appears normal. The tricuspid valve appears normal. No pulmonary artery drains it but the truncus arteriosus instead. Beneath the right posterior leaflet of the aortic valve is an 8 mm. opening into the left ventricle.

The right auricle appears essentially normal aside from its large size. It is in communication with the left auricle by a patent foramen ovale, 8 mm. in diameter.

The left ventricle is hypertrophied, but not nearly so much as the right; ventrically it measures 35 mm.; laterally, about 15 mm.; thickness of its wall in middle portion, 5 mm. No vessel leads from it, but it is in communication with the right ventricle by an 8 mm. opening occurring directly beneath the right posterior leaflet of the aortic valve at about the point where the aorta should normally arise. The mitral valve appears normal.

The left auricle is essentially normal. It has four openings for pulmonary veins into it.

The ascending portion of the truncus arteriosus resembles the normal aorta and from it arise two normal coronary arteries. The width of the truncus at its origin is about 20 mm. The first part of the arch is sacculated and its width is about 27 mm. From the summit of the arch the usual innominate and left carotid and subclavian arteries appear to arise. The descending portion of truncus arteriosus gives off ventrally two relatively large branches, hypertrophied bronchial arteries, the more superior leading to the right lung and the more inferior to the left lung. The usual small intercostal arteries are present.

The relationships of the various parts of the heart and vessels are shown in Fig. 3.

Abdominal viscera show no essential abnormalities.

The clinical symptoms are readily accounted for in view of the pathological findings. The greatly hypertrophied heart, in large part filling the thoracic cavity, pressed upon the esophagus and interfered with swallowing, nursing and nutrition. The lungs restricted in space by pressure of the much enlarged heart prevented complete aeration of the blood. Further aeration of the blood was also interfered with by the inadequacy of two hypertrophied bronchial arteries from the truncus arteriosus, acting for the lacking pulmonary artery and its branches. The hypertrophied bronchial arteries are not relatively as large as the bronchial arteries pictured in the adult case reported by Zimmerman.[2]

SUMMARY

A case of the rare anomaly, persistent truncus arteriosus, is recorded. The heart was enormously hypertrophied. Dysphagia was the main symptom calling attention to the defect. Necropsy revealed a persistent truncus arteriosus, and an opening from the left ventricle into the right ventricle. Enlarged bronchial arteries functioned as pulmonary vessels. Death occurred at eleven days of age.

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CONGENITAL DEXTROCARDIA WITH SITUS TRANSVERSUS COMPLICATED BY HYPERTENSIVE HEART DISEASE; ELECTROCARDIOGRAPHIC CHANGES*

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THAT congenital dextrocardia with complete situs transversus produces characteristic electrocardiographic alterations is well known. These changes consist of reversal in direction of all the waves in Lead I.[2, 3, 4, 5, 6, 7, 8, 9] The other leads remain unchanged. The reversal in direction of the waves results from the complete transposition of the anatomical and the electrical axis of the heart. This peculiar congenital anomaly does not interfere with normal life, and is frequently discovered accidentally in the course of a general examination. It is a curious fact that the electrocardiogram is not altered unless the malposition of the heart is accompanied by transposition of the abdominal viscera. This indicates, however, that with complete situs transversus, the axis of the heart is sufficiently altered to bring about reversal of the action currents of the heart, and the graphic inscription is reversed in Lead I, which, in general, represents the activity of the left side of the heart.

In persons who possess this anomaly, cardiac diseases of various types may develop as well as in perfectly normal persons. It is apparently unusual for superimposed cardiac disease to cause alterations in the already abnormal Lead I of the electrocardiogram. Such alterations did occur in the case reported here.

REPORT OF CASE

A man, aged 59 years, came to The Mayo Clinic because of weakness that had persisted after the occurrence of cerebral thrombosis four years previously. The only significant fact in the family history was that the patient's father had died at the age of 78 years, from cerebral hemorrhage. The patient had had scarlet fever at the age of 6 years, typhoid fever as a young man, frequent tonsillitis about 23 years prior to examination, mild pleuritis, and malaria. He had suffered from migraine for many years. He apparently had possessed good health until 4 years before he came to the clinic, when weakness suddenly had developed in the right leg and arm, while he was driving his car. There had been no loss of consciousness and no difficulty in speech, and he had been able to drive the car home. The function of the right leg and arm had improved rapidly but never had returned to normal. A physician had made a diagnosis of high blood pressure. Thereafter there had been persistent weakness, difficulty in mental concentration at times, peculiar behavior at intervals, and rather thick speech occasionally. Moderate exertion had caused the patient to become somewhat breathless, but there had been no evidence of congestive heart failure. There were no further complaints of significance.

*From the Section on Cardiology, The Mayo Clinic.

The patient was slightly overweight. He appeared older than his age. The teeth showed evidence of much dental work and the mouth was reddened. The peripheral arteries were moderately thickened. Cardiac dullnss was found to extend to the right, and the apex beat was visible just beyond the right nipple. The total area of cardiac dullness was definitely increased beyond normal. There was a rough, blowing, systolic, basal murmur. The systolic blood pressure ranged from 230 to 262 mm. of mercury, and the diastolic pressure from 140 to 142 mm. There was moderate (graded 2) sclerosis of the retinal arteries, with moderate retinitis of the type of severe benign hypertension. There was also thrombosis of a small inferior vein of the right retina, with hemorrhage. A detailed neurological examination disclosed arteriosclerosis of the vessels of the central nervous system and residual evidence of previous cerebral vascular interference. The blood count was normal, and there was only a faint trace of albumin in the urine. The Wassermann

Fig. 1.—Position of the heart, in a case of situs transversus.

test of the blood was negative. There were 32 mg. of urine in each 100 c.c. of blood. Roentgenographic examination of the thorax verified the presence of dextrocardia (Fig. 1); the heart was enlarged and the aorta was tortuous and slightly dilated. Fluoroscopy of the gastrointestinal tract revealed that the organs were transposed. Electrocardiography revealed sinus rhythm and reversal of the R- and P-waves in Lead I. The T-wave was upright.

The diagnosis was congenital dextrocardia with complete situs transversus, hypertensive heart disease, severe benign hypertension, artero-sclerosis of the vessels of the central nervous system and the peripheral arteries, and residual evidence of previous cerebral thrombosis.

COMMENT

The interesting and unusual feature of this case was revealed by the electrocardiogram (Fig. 2). As previously stated, congenital dextrocardia with complete situs transversus is always associated with

complete reversal of all the waves in Lead I. Here, however, the T-wave in Lead I was upright. Barnes and Whitten, and I[10, 11] are among those who have shown the frequent occurrence of T-wave negativity in Lead I of the electrocardiogram with hypertensive heart disease. Barnes and Whitten clearly showed that the inversion of the T-wave in Lead I, or in combined Leads I and II, occurred with unusual uniformity among patients who gave evidence of strain predominantly of the left ventricle. This interpretation is exclusive of myocardial infarction, in which different and additional factors are at work. In this case, in which inversion of the T-wave in Lead I was

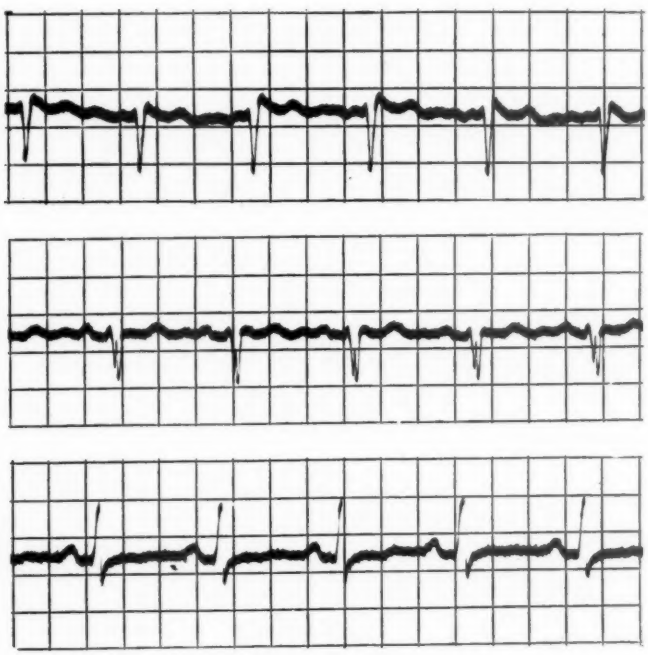

Fig. 2.—Electrocardiograms in a case of situs inversus.

anticipated as part of the disturbance attending congenital malposition of the heart, it was upright. Undoubtedly the upright position of the T-wave in this case represented the counterpart of T-wave negativity under conditions in which the heart is normally placed. It was, therefore, evidence of strain predominantly of the left ventricle, resulting from the severe hypertension.

The coexistence of congenital dextrocardia with complete situs transversus and acquired cardiac disease, in relation to additional changes in Lead I of the electrocardiogram is interesting, and study of additional cases unquestionably will reveal other unusual observations.

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CALCIFIED PLAQUE OF THE AORTA AT THE ENTRANCE OF A PATENT DUCTUS ARTERIOSUS: A POINT IN DIAGNOSIS*

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CASE REPORT

E. M., a colored woman, aged 33 years, was admitted to the Jefferson Hospital, May 20, 1931. She complained of pain in the right chest and shortness of breath.

The family history was negative.

Past history.—Pelvic inflammatory disease in 1925 and again in 1930; operated upon in 1925 and 1926. The patient had five living children and two miscarriages.

The present illness began about five weeks before admission with fever and general malaise followed by slight cough, then severe chills and high fever. Pain in the right side and increasing shortness of breath occurred just before admission.

Physical examination showed a poorly nourished young colored woman with some dyspnea, no clubbing of the fingers and no evidence of petechiae. The pulse rate was 120 and regular. The pupils reacted to light and in accommodation. The heart; apex beat was forceful in the 6th interspace at the anterior axillary line; left ventricle dulness apparently extended into the mid-axilla. The first sound had poor tone and there was a slight systolic murmur at the apex, also a rough systolic murmur over the pulmonary area. No thrill was felt. The right ventricle apparently was not extended. The lungs were resonant with the exception of the right base posteriorly and lower right axilla, where there was slight impairment of the percussion note but no râles. The abdomen was held rather rigidly. No organs or masses were made out. There was no edema of the legs. The knee jerks were active. About 5 c.c. of slightly turbid fluid were withdrawn from the base of the right chest posteriorly. No more could be obtained. No organisms were obtained from the fluid. The blood count on admission was hemoglobin 52 per cent, R.B.C.—2,940,000, W.B.C.—15,200 with 81 per cent polynuclears. On May 29 the W.B.C. were 27,000, on June 2, 31,000. The blood Wassermann was 4 plus. The blood culture was negative. The sputum was negative for tubercle bacilli. The urine contained a trace of albumin and an excessive number of white blood cells. The eye-ground examination was negative. Spinal fluid examination was negative.

Electrocardiogram.—Rate 110; rhythm regular; spreading of the base of the R-spike suggested delay of conduction along a branch of the bundle; a-v conduction time was normal; there was no disturbance of the muscle balance. Evidence of myocardial degeneration, probably advanced, was present.

X-ray of the chest showed no special pulmonary findings but an interesting observation was made regarding the heart and aorta, as follows: "There is an atheromatous plaque in the aortic knob and a slight fullness in the region of the left auricle. The heart is enlarged, particularly to the left. Its transverse diameter is 15 cm.; while that of the chest is 27 cm."

The patient was very ill and stuporous almost from the beginning. Near the end of the illness pulmonary findings became a little more marked, that is, dulness increased at the right base posteriorly, breath sounds were harsh, and numerous râles were heard. The temperature averaged about 103° with sharp peaks; the pulse and respirations accordingly were raised. Death occurred on June 7.

Autopsy was delayed until the following day. Interest centered chiefly about the heart which weighed 350 gm. The pericardial sac contained a little excess of cloudy fluid and there was some evidence of acute inflammation. The muscle of the

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left ventricle was soft and pale. A ductus arteriosus was present and was filled with recent thrombus material which stopped abruptly at the aortic opening of the ductus but continued on the pulmonic side so that a large soft thrombotic mass 1.5 cm. in diameter was attached to the wall of the pulmonary artery and projected into the lumen of the vessel. At the point of attachment of the ductus to the aorta there was a hollowed-out depression measuring 1 cm. in diameter. At this point was a large calcified plaque. It was flat and disk-like with very small nodular projections.

The right lung contained a number of suppurative areas varying in size from a few millimeters to several centimeters. The uterus, tubes and ovaries were matted together by dense adhesions. The tubes were dilated and the lumen of the left was closed off at its point of entrance into the uterus and several places in its course. In other places it was filled with bloody exudate. The left ovary and part of the tube were necrotic and there was considerable fibrous tissue reaction about them.

The post-mortem diagnoses were: (1) Bacterial endocarditis of the ductus arteriosus and adjacent pulmonary artery, (2) Calcified plaque of the aorta at the point of entrance of the ductus arteriosus, (3) Acute fibrinous pericarditis, (4) Multiple pulmonary abscesses, right lung, (5) Chronic suppurative left oophoritis and salpingitis.

COMMENT

The diagnosis of patent ductus arteriosus frequently can be made by the presence of a "machinery" murmur and intense thrill in the pulmonary area and evidence of an exaggerated pulmonary arc under the fluoroscope or on the x-ray plate. The absence of cyanosis and clubbing are negative points of importance in differentiating from congenital lesions of the pulmonary orifice. In the present case evidences of sepsis dominated the clinical picture and the murmur in the pulmonary area was considered to be of hemic origin. The calcified plaque of the aorta had a significance in this young woman which was not appreciated at the time.

A communication from Dr. M. E. Abbott, whose contributions to congenital heart disease are authoritative, informs me that in her series of 90 cases of patent ductus arteriosus calcific deposits at the aortic end of the ductus were observed in two cases, as follow. Murray[1] noted calcification of the wall of the aortic side of a patent and infected ductus in a patient aged 36 years, and White[2] recorded marked sclerosis and calcification at the aortic opening of a patent ductus arteriosus in a patient who attained the age of 66 years. However in White's case there was marked calcification of the remainder of the aorta. Abbott observes that the finding is probably more common than her series indicates. It is suggested therefore that in cases of suspected ductus arteriosus in young persons, in whom the presence of calcification from ordinary arteriosclerosis would be unlikely, roentgen-ray evidence of calcification in the arch of the aorta may be looked upon as a confirmatory sign.

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Society Transactions

AMERICAN HEART ASSOCIATION

SEVENTH ANNUAL SCIENTIFIC SESSION

JUNE 9, 1931

The morning session was called to order by the President, Dr. Robert H. Halsey of New York, in the auditorium of the Institute for Mental Hygiene of the Pennsylvania Hospital, Philadelphia, Pa., at 9:40 o'clock. The following program was presented:

1. DR. R. L. HAMILTON, SAYRE, PA.—**Precordial Pain—Its Cause and Significance.** (Abstract of Dr. Hamilton's paper.)

The increasing incidence of anginal pain and of sudden death has focussed the attention of the public as well as of the physician on precordial pain. Two hundred case reports of patients with precordial pain associated with organic or functional cardiac disturbances were analyzed. In sifting out the cases 27 different extracardiac causes of pain were encountered. The cases with pain associated with cardiac disturbances could be separated into five groups: (1) those with relative coronary insufficiency, (2) those with aortic pain, (3) those with pain produced by a viscerosensory reflex, usually from the gall bladder, (4) those with acute pericarditis, and (5) those with pain associated with an increase in venous pressure.

The most common diagnoses were coronary occlusion and angina pectoris. The principal etiological factor was infection between the ages of thirty and forty years, hypertension between the ages of forty and fifty-five years, and arteriosclerosis in patients more than fifty-five years of age. The prognosis was uncertain, especially in the middle group, but in general was better in the younger patients and in the very old. There were more cases of organic heart disease in men than in women, although the incidence of hypertension was nearly the same in the two sexes, and the incidence of neuroses was higher in women. The study suggested that heredity, sedentary occupation with mental strain and possibly the use of alcohol and tobacco played a part in the production of precordial pain. The incidence of infections and of associated diseases agreed with the findings of other workers.

2. DR. FRED M. SMITH, IOWA CITY, IOWA.—**Presentation of Case.** (Abstract of Dr. Smith's presentation.)

Dr. Smith presented the case of a man, aged sixty years, who gave a characteristic history of angina pectoris. The distress began over the lower substernal region and was transmitted to the left shoulder and often down the left arm. It was invariably precipitated by exertion or excitement. He first came under the observation of Doctor Stroud three years ago shortly after the initial attack which lasted about thirty minutes. During this period he had experienced numerous attacks of varying intensity. There was a slight increase in the size of the heart. Otherwise

the cardiac findings were essentially negative. The blood pressure was well within normal range, and there was no history of hypertension. The blood Wassermann was negative. The urine likewise was negative.

The patient was of particular interest because of the relationship between angina pectoris and coronary occlusion. Each of these conditions was ordinarily associated with an advanced sclerosis of the coronary arteries. They probably represented different steps in the same pathological process and differed only in the extent of the functional impairment of the myocardium. Coronary occlusion might be regarded as a complication of coronary artery disease, and it was not surprising that it should occur during the course of or terminate in angina pectoris. The history of the sudden onset of a rather severe form of angina pectoris in this patient and the duration of the initial attack suggested a coronary occlusion. The electrocardiogram taken some time later showed suggestive changes in the T-wave which supported this possibility. It was generally known that coronary occlusion might precipitate an angina pectoris. This means of producing an angina was, however, probably far more common than was generally recognized. It was furthermore not improbable that minor forms of coronary closure were frequently overlooked during the course of angina pectoris.

3. Dr. Alfred Friedlander, Cincinnati, Ohio.—**Electrocardiograms in Patients With Cardiovascular Lues as Compared With Electrocardiograms in Cardiovascular Disease of Other Etiology.** (For original article, see page 15.)

4. Dr. Lewis A. Conner, New York, N. Y.—**Presentation of Case.** (Abstract of Dr. Conner's presentation.)

Dr. Conner presented the case of a man of seventy years who for several years had had attacks of anginal pain of increasing frequency and severity. At first the attacks came on only on walking, later they appeared also with excitement, after eating and sometimes during the night. There had been no attacks that could be recognized as those of coronary thrombosis. The speaker discussed the case only from the standpoint of treatment. In his own experience treatment of effort angina by drugs and by rest had given such very disappointing results that he had been led to seek some other method of treatment. He called attention to the fact that different forms of physical effort vary greatly in their proneness to induce anginal pain; many patients who could not walk even a short distance without the appearance of pain were yet able to take other forms of active exercise, such as running upstairs or calisthenics, without such pain. In view of the evidence offered by Gross as to the pronounced tendency toward increased vascularization of the heart muscle in the middle and later years of life, it seemed rational to attempt to hasten the development of new coronary branches by the use of graded exercises, provided such exercises could be carried out without inducing pain. For the past two or three years he had been using such graded exercises, although at first very hesitatingly and cautiously. The early experiments had been sufficiently encouraging to warrant the systematic use of such exercises, and the results had thus far been highly satisfactory. Only a few cases, however, had been followed sufficiently long to know if the benefit was likely to be permament. One case had been followed two years. At the time these exercises were started this man had been unable to walk at all because of the severe pain and had been confined to his room. After a month or two of graded exercises the pain had so far disappeared that he could resume active work, and for the past year he had been free from pain and actively engaged in his work as civil engineer. In several other cases there had been freedom from

pain for a number of months. He had never seen a case in which he thought harm had been done by the exercises. He had used the setting-up exercises of the Army, consisting of light, quick movements, limiting them at first to four or five minutes and having them done several times a day, preferably every two or three hours. It was important that they should be such as did not bring on pain, that they should not be done immediately after a meal and that they should be carried out in a comfortably warm room, in other words that the conditions likely to induce anginal pain be avoided as far as possible.

There was another, more serious aspect of these cases of effort angina, which was the likelihood sooner or later of an attack of thrombosis. If it was possible by graded exercises to increase the anastomotic circulation in the heart, it seemed reasonable to suppose that this increase might lessen materially the serious effects of such a closure if it should come.

The speaker said that the details of the method of treatment offered were by no means firmly established and could probably be improved upon, but he was quite convinced that if the problem were ever to be solved the solution would come from some method of development of the anastomotic circulation in the diseased coronaries.

5. Dr. Drew Luten, St. Louis, Mo.—**Contributory Factors in Coronary Occlusion.** (For original article, see page 36.)

6. Dr. Gordon E. Hein, San Francisco, Calif.—**Presentation of Case.** (Abstract of Dr. Hein's presentation.)

Dr. Hein presented the record of a young man of twenty-nine years who had an attack which resembled one of coronary thrombosis and who died thirty-six hours later. The diagnoses suggested were: (1) coronary infarction; (2) rupture of the aorta, possibly secondary to aneurysm of the ascending aorta; and (3) occlusion of a large branch of the pulmonary artery. Autopsy revealed narrowing of the isthmus with dilatation proximal to this and a transverse tear 2 cm. long 6 cm. above the aortic cusps.

Dr. Hein discussed the recognition of coaretation of the aorta in those cases in which the diagnosis is not easy and commented on the relative frequency of rupture of the aorta in this condition.

7. Dr. Stewart R. Roberts, Atlanta, Ga.—**Nervous and Mental Factors in Angina Pectoris.** (For original article, see page 21.)

The afternoon session was called to order at 2 o'clock, and the following program was presented:

8. Dr. John H. Musser, New Orleans, La.—**The Familial Incidence of Coronary Thrombosis.** (For original article, see page 45.)

9. Dr. David Riesman, Philadelphia, Pa.—**Presentation of Cases.** (Abstract of Dr. Riesman's presentation.)

Dr. Riesman showed a little Italian girl of thirteen years who exhibited in a striking manner Broadbent's sign of retraction of the lower chest, laterally and posteriorly, as the result of adherent pericardium. The child had suffered for years from rheumatic pancarditis—both aortic and mitral valves were involved; the heart was greatly hypertrophied; retraction of the ribs had steadily increased during the

year and a half she had been under observation. When first admitted her tonsils had been found large and infected, the glands at the angles of the jaw and in the neck were likewise much enlarged. Despite her serious cardiac disease, she bore tonsillectomy under general anesthesia as well as a healthy child. The glands of the neck promptly diminished in size after the operation and soon could not be felt. Taking out the tonsils in this case was, of course, like shutting the stable door after the horse had been stolen.

Dr. Riesman passed around Broadbent's little book in which the sign known by his name was described.

The second patient was a colored man who had recently been admitted to the Philadelphia General Hospital in an advanced stage of circulatory decompensation. Under tapping and novasurol the signs of decompensation had rapidly disappeared. Dr. Riesman presented him as an example of nonvalvular heart disease. There was nothing in any way abnormal audible on auscultation of the heart; the blood pressure was about normal—it might have been higher previously, but of that there was no information; the Wassermann test was negative; there was no evidence of renal disease. The speaker discussed the nature of such cases of decompensation which evidently were due to muscular and not to valvular defect. He believed that coronary disease might be the basis of the cardiac myasthenia. At autopsy such cases showed no signs of inflammation. Hence the clinical term myocarditis was misapplied; he had coined the term "myocardosis" to designate the condition.

Dr. Riesman also spoke of the increasing frequency of myocardial failure since the onset of unemployment and hard times and was inclined to attribute it to food deficiency.

The ultimate cause of myocardial failure was probably chemical, whether faulty metabolism of lactic acid or something else was still undetermined.

Attention was called to the frequency with which hydrothorax is overlooked. The striking effects of novasurol were also pointed out.

10. Dr. Joseph T. Wearn, Cleveland, Ohio.—**The Relationship of the Thebesian Circulation to Coronary Occlusion.** (Abstract of Dr. Wearn's paper.)

Correlation of clinical findings with the necropsy reports has shown many cases which revealed few signs or symptoms referable to the heart and yet whose coronary arteries were practically closed. Arteriosclerosis was the most common cause of the occlusion. Syphilitic aortitis, in some instances, resulted in the complete closure of both coronary orifices and in many instances occluded the opening of a single coronary artery.

These processes—arteriosclerosis and syphilitic aortitis—bring about a gradual occlusion of the coronary arteries, and in many instances they produce few, if any, distressing signs or symptoms of heart failure. Indeed, some escape detection during life.

Such cases are in distinct contrast to those where there is a sudden occlusion of a branch of a coronary. In these heart failure is usually the immediate result and death frequently follows.

When the coronary arteries are completely occluded either at their mouths by syphilis or in their main trunks by sclerosis, the hearts may carry on their work and eventually when examined they do not show infarction. These findings make it necessary to find another source of blood supply to the myocardium, and this blood supply must be sufficient to enable it to maintain a normal circulation.

Batson and Bellet[1] have suggested a reverse flow from the right auricle through the coronary sinus and great veins as a means of blood supply to the myocardium.

That there might, under the conditions of their experiments, be a slight back flow into the veins during auricular systole is quite possible. But their suggestion that the low pressure in the right auricle can maintain a circulation in the myocardium sufficient to enable it to keep up a normal blood pressure, is to me not acceptable.

Histologic studies[2] have shown direct communications between the Thebesian vessels in the ventricles and the capillaries of the myocardium. No valves have been found in the Thebesian vessels. The blood under ventricular pressure, therefore, has direct access to the capillary bed of the heart.

It is suggested that in many of the patients whose coronary arteries become occluded, the Thebesian vessels offer a channel of communication between the ventricles and the capillaries.

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11. DR. JAMES B. HERRICK, CHICAGO, ILL.—**Presentation of Cases.** (Abstract of Dr. Herrick's paper.)

Dr. Herrick presented the following cases:

CASE 1. An adult woman after a prolonged stay in the hospital, with many weeks in bed, made a good recovery from what was clearly a moderately severe attack of coronary thrombosis.

Comment: Two points may be emphasized, the importance of rest in the treatment, and the difficulties in the way of diagnosis as well as of treatment when, as in this case, there were exaggerated nervous symptoms of a functional character.

CASE 2. A white man of forty-four years with a history of chancre at seventeen years, was suddenly overcome by pain in the chest, right shoulder, face, epigastrium. He was dizzy and for a few minutes may have been unconscious. Taken at once to a hospital, he complained of a dull ache in the precordium and between the shoulder blades. He was not dyspneic or cyanotic. The heart tones were faint, the pulse was just perceptible. There were many extrasystoles. On one day, for a few hours only, a pericardial friction sound was heard. The temperature rose to 100.6°. There were 11,200 leucocytes. An electrocardiogram taken on the ninth day showed QRS complexes of low amplitude and notched in all leads. The T-wave was inverted in Lead III. Three weeks later typical coronary waves were seen in Leads II and III. The diagnosis was acute coronary occlusion. It is now two years since the accident. He is able to do light work with only slight fatigue and slight shortness of breath. He complains at times of mild precordial distress and palpitation.

Comment: One may note the nearly complete functional recovery after rest. While syphilis in this case may have been a factor contributing to the occurrence of the thrombosis, no definite proof was afforded of relief from specific remedies.

One must be prepared to recognize cases of coronary thrombosis with mild or atypical symptoms. Often diagnosis must be made not on any one symptom but on the grouping of symptoms, the instrumental findings and the exclusion of other diseases. Of especial value in this case were the fever, leucocytosis, pericardial friction sound and the extrasystoles. The low voltage and notched curves were also suggestive. Still more significant was the fact that three weeks later the electrocardiogram showed further changes. The process was clearly one in which the myocardium was undergoing alterations presumably in the way of healing. The especial point to be stressed is that in these acute cases the tracings are not always fixed and unalterable. The diagnostic value of *change* in the electrocardiogram should not be overlooked.

12. Dr. Paul D. White, Boston, Mass.—**The Prognosis of Angina Pectoris (500 Cases) and Coronary Thrombosis (200 Cases).** (For original article, see page 1.)

13. Dr. Emanuel Libman, New York, N. Y.—**Presentation of Case.**

DISCUSSION

Dr. Wolffe, Philadelphia, Pa.—Dr. Hamilton's paper is of great importance because of his clear observations. Numerous statistical studies have been reported, but it would be of great benefit to the students of angina pectoris if we were to have more studies of the character presented here this morning. The etiology of angina pectoris, as Dr. Hamilton pointed out, is still unknown although many theories have already been advanced. The clinical and pathological manifestations have been pointed out and are fairly well recognized. May we not draw some conclusions from the work already done? If one were to consider all these manifestations, as well as some of the known pathological lesions associated with this malady, a rather important conclusion would suggest itself. May not all the various etiological factors produce this sympton complex by a common *modus operandi?* May we not look upon angina pectoris as a reflex due to an imbalance of the two opposing parts of the autonomic nervous system, a parasympathetic and sympathetic imbalance (although even this nomenclature may have to be somewhat modified)? The symptoms and physical signs may depend greatly upon the preponderance of one or the other side of the autonomic nervous system. The drugs to be employed to check this dangerous and painful complex will also depend on the proper recognition of this disturbed balance. Dr. Hamilton clearly showed how the heart beat returned and the volume improved following the administration of epinephrin, yet Levine and his coworkers showed that epinephrin will in many cases precipitate an attack of angina pectoris. This has also been the experience of many clinicians. It is also a common experience that the same type of therapy given for a similar condition will improve one patient and aggravate the symptoms in another. May it not be well carefully to investigate symptoms and physical signs and see which side of the autonomic nervous system has the upper hand and direct treatment accordingly—using "inhibitor" drugs for the "accelerator" type and "accelerators" for the "inhibitor" types? This thought may be applied to many other diseases. The T-wave appears to be a very important guide provided we do not speak of it as a "coronary T-wave" but rather as a T-wave of sympathetic and parasympathetic imbalance.

Dr. Hamilton, Sayre, Pa.—With regard to Dr. Wolffe's observations with adrenalin I might say that not long ago I had a patient who I thought had a paroxysmal hypertension and gave him a little adrenalin to see whether or not I could bring out the symptoms. He developed such an intense coronary pain that for a few moments it seemed as though he might die.

Dr. Oppenheimer, New York, N.Y.—Dr. Friedlander has mentioned incidentally the work of Dr. Master on certain electrocardiograms found in cases of hypertension, i.e., left axis deviation associated with inversion of the T-wave in Lead I, or sometimes in both Leads I and II. At the time of publication there was some doubt whether such T-wave changes were not due to coronary artery occlusion complicating hypertension. Since then I have had the opportunity of following a few such cases and seeing the hearts at necropsy, and have been convinced that such electrocardiographic changes may occur without coronary occlusion. For example, one patient with marked essential hypertension in whom these typical electrocardiographic changes persisted up to the day of his death, showed at necropsy an enormously

hypertrophied heart, weighing 1050 grams, and yet no coronary occlusion or myocardial infarction to account for his T-wave changes. Similarly electrocardiographic changes have been seen in instances of rheumatic aortic valvular disease, without finding coronary occlusion in the heart at post-mortem examination. So one may say that such inversions of the T-wave in the presence of left axis deviation do not necessarily mean coronary artery occlusion.

DR. PARDEE, New York, N. Y.—I should like to say a word about the relation of electrocardiographic abnormalities to mortality and to prognosis. In this table and in many other similiar studies an effort has been made to decide whether or not certain peculiar features of the electrocardiogram have some relation to the length of time a patient is going to live. It seems to me that in order to have such a decision of any value, the figures must point overwhelmingly in one direction, that is, the percentage must be as high as 80 or 90, because there are so many other factors besides the electrocardiographic abnormality that must be taken into consideration in the prognosis. I do not feel that any of the percentages in Dr. Friedlander's tables indicate a definite seriousness of the observed physical sign. Electrocardiographic abnormalities are only a small part of the clinical picture, and I think that this is the reason that different authors, in attempting to estimate their importance, have found varying percentages and different trends. To me the figures would have to point much more strongly in one direction than do these before they would have prognostic significance.

DR. WOLFFE, Philadelphia, Pa.—Dr. Friedlander's paper again proves that there is no such thing as a pathognomonic coronary T-wave. This type of T deformity may be produced as have already pointed out if the animal's autonomic mechanism is disturbed by administering certain drugs and particularly certain hormones. A coronary insult may also produce a similar T-wave, but apparently due to a similar mechanism. It seems that if the coronary insult is superficial it involves one side of the balance, while if deep, the other.

The T-wave promises to be a very important index in diagnosis and treatment in every branch of medicine. It would be difficult in these few minutes to offer enough proof even to suggest changing deeply rooted ideas. I can nevertheless assure you that we have records showing typical coronary T-waves in individuals with a perfectly normal cardiovascular system as far as organic disease is concerned. In the dog we can depress the T-wave by the use of the substance I previously mentioned as desympatone and bring it up again by administering epinephrine.

I may say in conclusion that those working with the electrocardiograph should keep in mind parasympathetic and sympathetic imbalance, whether due to coronary disease, a blow over the chest or epigastrium, a sensitized nervous system, or gall bladder disease; for all may produce changes of the T-wave, and therefore a diagnosis and prognosis should not be based entirely upon this finding.

DR. MARVIN, New Haven, Conn.—Dr. Conner has spoken of the work of Gross as indicating that as an individual grows older he develops fairly abundant anastomoses between the branches of the coronary arteries. It seems to me of some importance to indicate that this view of Gross' is by no means generally accepted. I have discussed the matter with some pathologists who have no hesitation in regarding it as being incorrect, unsound in theory and untrue in fact, and they point out that if this is true of the heart, it is peculiar to the heart muscle, that such processes do not occur in other tissues of the body as the individual grows older, that this process is a development of an abundant anastomosis of new, young and growing tissue and not one that is undergoing degenerative processes. Those of you who have seen the illustrations in Gross' book will agree that they

are apparently conclusive, but there is an alternate explanation which seems to some of us acceptable. It is this: in the younger hearts the arteries are more elastic, either they do not permit the injection medium to enter them because they are closed by virtue of their elasticity, or, having permitted the injection medium to enter, they probably squeeze it out into the larger vessels so that if x-rays of such hearts are taken no anastomoses are demonstrated. As the hearts grow older the arteries become more sclerotic, less elastic, permit the injection fluid to enter, and the apparent anastomoses are shown which may actually have been present for many years.

DR. LIBMAN, New York, N. Y.—There is no reason why a collateral circulation should not gradually develop, in view of the studies of Kusnetzowsky and others that the coronary arteries begin to show changes from birth on.

DR. THAYER, Baltimore, Md.—I have been very much interested in what Dr. Conner has said. I have been much interested in the subject in general, and it seems to me that there are a good many sides to it. In the first place, if one wants to help an individual who has angina of effort, such as the patient of Dr. Conner, it seems to me the first thing one can do is to let the patient know what is the matter with him. That does not mean to tell him he has angina, of course, that would be a stupid thing to do, but it does mean making friends with the man and telling him as well as you can what you think is the matter with his circulation. Then it seems to me the next thing to do is to encourage him to feel that he can do certain things to improve his circulation very much. If I can, I usually put him to bed for a certain period but this is never done without combining it with massage. Now generally when it is done, after a certain length of time the patient becomes greatly encouraged to find that he is going through his days without the pain, and then, after a certain length of time, I always begin—perhaps not quite so vigorously as Dr. Conner has—but I always begin with the setting-up exercises that were used in the U. S. General Hospital No. 9 for the treatment of the so-called effort syndrome. I have often been quite amazed at the improvement one may get. I have in mind the case of a man of seventy who had been having five or six attacks every night, besides great difficulty during the day. As he would not go to the hospital, he was put to bed at home and later was put on the graded exercises. The old fellow went a year or two actually without pain and has been able to continue at his work for three or four years. He learned, as these people do, that he could get rid of his pains to a very considerable extent if he would regulate his life. You can talk to a man for days and accomplish nothing, but a demonstration teaches him how to live just as Weir Mitchell taught neurotics how to live by giving them a rest cure.

DR. LEE, Washington, D. C.—May I ask Dr. Conner if he includes in these exercises cases of previous coronary thrombosis, and if so, after what general period of rest time?

DR. MCCULLOCH, Saint Louis, Mo.—Dr. Conner's presentation of this case has brought out some ideas that are extremely important, particularly the differentiation between sustained effort and the slight effort of short duration in producing pain. It has been pointed out recently by Bramwell and Ellis* that individuals with normal hearts may respond to different forms of exercise in various ways. As a result of their studies on runners in Olympic Games they state that sprinters who run relatively short distances can do so without breathing or using the heart during the time they are undergoing this form of exercise; whereas, a runner who does sustained effort, for example, during a marathon race will have a very different re-

*Quarterly J. Med. 24: 329, 1931.

sponse to this exercise; that is, the respiration, circulation and heart muscle must be so conditioned as to carry on during the longer period of excessive effort. The circulatory conditions must be such that the heart muscle receives an adequate supply of food material and an adequate removal of waste during this period. It may be that this is of some importance in allowing exercise for such patients as Dr. Conner describes because the circulatory needs of a patient with coronary thrombosis or with interference in coronary flow from other causes may be comparable to the person undergoing a marathon race; whereas, the patient with obstructed flow through a heart valve may be comparable to a person undergoing a short sprinting exercise. It seems to me, therefore, that the differentiation between these two forms of exercise is important and should be kept in mind as Dr. Conner has pointed out. The explanation that might be offered for the difference in the response between the two exercises is that the reserve in the heart as well as the skeletal muscle cells may be used up completely in sustained effort without adequate provision for replenishment of materials or removal of waste through the injured coronary circulation.

DR. CONNER, New York, N. Y.—I do not feel qualified at all to discuss the point raised by Dr. Marvin concerning Gross' conclusions. I know there is a difference of opinion and I am quite prepared to believe that such increase in vascularization may not be a universal thing, but it seems to me one cannot deny that the heart is capable of developing an elaborate anastomotic supply, otherwise I do not see how one can explain these cases that one sees occasionally, such as those that Dr. Wearn has reported upon, of complete or almost complete occlusion of both coronaries and yet with the maintenance of fairly good health. There must be an elaborate anastomotic supply, as well as the increased use of the Thebesian vessels I should think. I am much interested to hear what Dr. Thayer has to say about the use of exercises. I have not attempted to discuss the many collateral things in the treatment, but, of course, I agree fully with Dr. Thayer as to the need of having the patients—if they are intelligent at all—understand the circumstances and understand the need of living within the limits of their capacity; but I am glad to know that he has been trying to work out in the same way that I have this matter of exercise. I neglected to say that I have felt that the selection of suitable cases was very important, and at the beginning I should not have thought that this man of seventy with evident myocardial change was a very suitable case, but as I have gone on, I have come to feel, apparently as Dr. Thayer feels, that even cases that have definite and serious myocardial damage can still be benefited by such exercise. To answer Dr. Lee's question, I have not used the exercise at all in those people who have had definite coronary thrombosis and who have the after-pain. I do not quite know the significance of the pains that come after a coronary thrombosis. I have been inclined to associate them with the actual damage done by the thrombosis. It is possible, I suppose, that they may be merely anginal pains from some other arterial branch or branches that are sclerotic and are beginning to give their symptoms, but usually these post-thrombotic pains do not have quite the typical behavior of those of effort angina.

DR. LIBMAN, NEW YORK, N. Y.—In connection with Dr. Luten's discussion of the subject of pain, I shall probably have time to indicate this afternoon that all of the proposed theories have truth in them but that they apply to different types of cases. I should like to emphasize the subject of contributory factors in coronary thrombosis. In 1919 I pointed out that patients who suffer from coronary thrombosis may have a history of thrombosis in veins, and are liable to develop thromboses in the arteries of the lower extremities and of the brain, often insidiously. I indicated that there was something present apart from the disease of the vessel wall—especially as thrombosis may occur in a coronary artery slightly diseased and

not in those showing marked changes. This condition I have since termed "thrombophilia." Recently I found that a writer by the name of Mandel used the term "thrombophilie" for the same purpose (original paper not yet located). It is important I believe to seek along the line of metabolic disturbances for an explanation of the larger number of cases of coronary thrombosis. If one studies their histories and also the family histories, one finds many conditions present which we group as gout, typical or atypical, or as "goutiness." In going over my records I find almost the same disturbances present as Allbutt found in his elaborate studies of families in Leeds, carried on for a long time. It is interesting to find that von Recklinghausen stated (not specifically in relation to the coronary arteries) that there exists a tendency to thrombosis in certain individuals, apart from circulatory factors, and that this is associated with metabolic disturbances such as gout and diabetes. A study of this subject should give us important clues in treatment and in prevention. I might add parenthetically that I am of the opinion that the factors that predispose to coronary thrombosis also play a rôle in the development of atherosclerosis of the coronary (and other) arteries and of the nerve type of so-called angina pectoris. This would explain why we may encounter these conditions, associated and separately.

Dr. Katz, Chicago, Ill.—In regard to Dr. Luten's paper, emphasis was laid on the fact that spasm or constriction occurs in the coronary arteries clinically. The idea was based on the work of Anrep and Segall, which is contradicted in part by Wiggers. Anrep and Segall computed the coronary inflow from the flow from the coronary sinus on the assumption that 60 per cent of the total inflow was measured by the coronary outflow, a fact which Wearn has questioned. I therefore wish to point out that there is no clear evidence that coronary spasm or constriction occurs, or, at least, that it is of any significance clinically in causing impediment to coronary flow. All the changes met with in coronary flow, aside from those caused by organic lesions, can be readily accounted for by changes in blood pressure—especially the diastolic. Anrep has shown clearly that the most important determining factor for coronary flow is the aortic blood pressure. Any marked lowering of the blood pressure will cause an ischemia of the heart and thus cause pain and other symptoms, and may, as in a case which Dr. Wallace and I reported, lead to death with the typical signs of coronary occlusion. There is no need to assume that coronary spasm occurs until direct evidence is brought forward for its occurrence, or until proof is given that changes in blood pressure cannot explain all the effects attributed to spasm.

Dr. Luten, Saint Louis, Mo.—I am very much obliged to Dr. Libman. His remarks go further back to ultimate causes than mine. Diabetes, of course, is particularly associated with coronary sclerosis in the first place, but even so, certain patients with coronary sclerosis experience occasions of low diastolic pressure without occlusion while others get occlusion under such circumstances. Dr. Libman's explanation would tend to account for this difference. I do not mean to imply that gastrointestinal reflexes in causing possible coronary constriction are the principal contributory factors in occlusion. Most cases of occlusion occur at night, particularly in the early hours of the morning, when diastolic pressure is low. I was merely offering evidence that such reflexes may constitute additional contributory factors. One bit of collateral evidence lies in the work of Pearcy and Howard. They produced extrasystoles and changes in the ventricular complex by visceral stimulation. It is common clinical experience, of course, for disturbances in the gastrointestinal tract to produce extrasystoles. Now, Pearcy and Howard are convinced that this is not due to pressure but to a reflex.

DR. THAYER, Baltimore, Md.—I should like to ask Dr. Hein what was the condition of the aorta in the region of the rupture, whether there were marked degenerative changes. As I understand there was no syphilis. It would be rather a striking thing if the aorta had ruptured in the absence of obvious anatomical change near the point of rupture.

DR. HEIN, San Francisco, Calif.—The aortic wall was smooth except just at the point of rupture. It was decidedly thin; there was no arteriosclerosis of the ascending aorta and no abnormality of the valves. Congenital weakness of the aorta may be one of the reasons for rupture, and in such cases apparently the rupture is within the first 6 or 8 cm. of the ascending aorta. We all know that coronary occlusion may occur without pain, but we are beginning to be a little suspicious of our diagnosis in those cases in which pain is not present. In the last two cases in which we made the diagnosis of coronary infarction in the absence of pain, our diagnosis was wrong.

DR. THAYER, Baltimore, Md.—I should like to ask Dr. Roberts if he is not able sometimes to extract from a colored patient, in whom he suspects angina, something which confirms his suspicions: a fairly clear story of angina? I do not know that I can remember ever getting it myself, but I think since we have been very much interested in the subject in Baltimore we have been able, when we suspected it from the general story, to get something in that direction. I remember one woman with coronary thrombosis; there was not one word in her history as written that suggested pain, but quite accidentally I happened to see the student who met the patient as she came to the out-patient department; he told me that she had complained of a toothache-like pain in her left forearm. And now and then I think one can get the equivalent of the story of angina from the patient. But angor animi and the clear objective description of the pains, one does not get.

DR. MARVIN, New Haven, Conn.—I should like to ask one general question which I hope several of the speakers will answer, and that is what their experience has been with the use of the xanthine derivatives, namely metaphyllin, theocin and theobromine, in the treatment of this condition. My own experience has been most discouraging; I am not satisfied that I have ever seen any benefit follow the use of any of these.

DR. OPPENHEIMER, New York, N. Y.—May I call attention to a certain parallelism between exophthalmic goiter and angina pectoris from the point of view of the rôle of nervous and mental influences as an etiological factor? I happen to be connected with a New York hospital which is in a neighborhood in which there has been a large influx of negro population and of about 100,000 Porto Ricans. Formerly Graves' syndrome was considered rare in the negroes in New York City, but of late such cases have ceased to be rare among the negresses, and we have even had two negroes with exophthalmic goiter in the ward at the same time. Graves' syndrome has also been observed recently in a few Porto Rican women. In both groups, negro and Porto Rican, I am under the impression that industrialization under urban conditions is responsible for the apparent increase of exophthalmic goiter. Is it not the instability of the whole nervous system, more particularly, however, of the autonomic nervous system, which is an etiologic factor in the apparent increase of Graves' syndrome and also of angina pectoris in the urban population? I should like to ask Dr. Roberts whether he has noted Graves' disease in the negro population in his neighborhood.

DR. CONNER, New York, N. Y.—I must say a few words about the interesting paper of Dr. Roberts', even though we may be a few minutes late. Almost the finest thing that comes from these meetings of ours are these frank and full dis-

cussions, and we must all have felt deeply the effect of Dr. Roberts' wise presentation of this whole subject. It is extraordinary that such differences as he points out should occur in the different races, but I have no doubt they do occur, although they are perhaps not quite so positive as we now think.

Now to go back to the question of the great increase in angina and in coronary thrombosis—I have no way of knowing, I do not think anybody knows, the real situation. We get the impression that they have increased and it may well be so, because we have had a great admixture of races and great changes in our civilization; but I venture to say that Heberden, when he first saw these cases and described them so beautifully, must have wondered, since no one had described them before, if the disease was not greatly on the increase, and if such increase was not a result of the strain and stress of life in 1768 as compared with that of a century before.

Much of the apparent increase recently must be ascribed, I think, to the fact that these conditions are now so widely recognized both in the profession and in the laity.

DR. HALSEY, New York, N. Y.—By way of proof of the rarity of the disease in early days I might state that the records in New York City would indicate that no death occurred from heart disease from 1804 to 1808.

DR. ROBERTS, Atlanta, Ga.—To begin with Dr. Marvin's question first I agree with the intimation in his question.

To answer Dr. Oppenheimer I think I have seen only one case of exophthalmic hyperthyroidism in a pure African. One should distinguish between the pure African and the mulatto—there is a vast difference, as Dublin, in his statistics shows, in regard to their susceptibilty to disease. I think your point is well taken, and that as the negro turns from the quiet country districts to the city with its industrialism we shall see the development of diseases in him that we have not heretofore seen. I do see occasional exophthalmic hyperthyroidism in mulattoes and, strange to say, adenomatous hyperthyroidism in my experience is particularly rare in the full-blooded African.

In regard to Dr. Thayer's point, I distinguished carefully in my remarks between the anginal symptoms of coronary thrombosis and angina as we know it without thrombosis or evidence of lues. We may have been not so thorough as we might in getting the history. A negro is an individual from whom it is very difficult to extract a correct history. But I certainly want to disagree with the statements that the negro is not as susceptible to pain as a white. In my experience he is more susceptible to pain than the white. But the point in my mind is that while he has a very well developed susceptibility to pain and sensitivity to pain he is lacking in the ability to worry unpleasantly. I was careful not to say that anginal pain does not occur in the full-blooded African, but to say only that we had not seen it. Such pain does occur in association with luetic aortitis. Although statistics are so unsatisfactory, I think the impression prevails, probably correctly, that angina is on the increase. I am sure that I have seen more cases of angina during the Florida "boom" and subsequent to its collapse than I ever saw before, and I think angina has been rather more prominent since a famous day in November, 1929, in New York City than it was before.

DR. RABINOWITZ, Brooklyn, N. Y.—The observation of Dr. Musser that in coronary disease the next generation suffers at a younger age is also borne out in the studies of cancer families that the cancer will occur at a much earlier age than it did in the preceding generation. The same observation has been made in cases of apoplexy. I have had an instance where the son died of an apoplectic stroke two years before the father.

DR. CONNER, New York, N. Y.—Mr. Chairman, speaking of arteriosclerosis, not just what Dr. Musser was emphasizing of course, I cannot resist the temptation to say that I think the habit of speaking of *general* arteriosclerosis has done more to confuse us and to cloud our understanding of the arteriosclerotic process than anything I know of, because arteriosclerosis is usually not general. It may be so ultimately in old age or with a long-standing hypertension, but arteriosclerosis—and you find such evidence all through the literature on the subject—is usually a local thing through all its earlier stages. Usually, too, it involves certain of the viscera, apparently often chiefly one, for a long while almost to the exclusion of the others and certainly often to the exclusion of the peripheral arteries. I do not know of anything more misleading than to attempt to appraise the state of the arterial system as a whole by running over a few peripheral arteries and judging it by their normal state or by their changes. It seems to me that it ought to be emphasized that arteriosclerosis is a focal thing to begin with, that in the viscera it often begins in very early life and may remain, as I have said, in one or a few viscera for a long time without any general involvement at all. Then I think with that conception we can understand how it is that these people at 35 or 38 or 40 years so often have their coronary attacks, just as others have cerebral attacks, and still others develop changes in the renal arteries at an unusually early age.

DR. KATZ, Chicago, Ill.—I should like to ask Dr. Wearn how the adjustments take place in the Thebesian vessels, whether this requires time, or whether the change occurs at the time the patient has a coronary occlusion.

DR. RABINOWITZ, Brooklyn, N. Y.—Has Dr. Wearn found greater enlargement of the thebesian vessels in cases of coronary occlusion than in apparently normal hearts?

DR. WOLFERTH, Philadelphia, Pa.—There are one or two points that might be raised here I think. Dr. Batson and Dr. Bellet, in maintaining a venous pressure at the normal level, were able to get particles of graphite deep into the coronary venous channels. Now, the only conclusion one can come to is that these particles had gotten down there by a reversal of flow, under the conditions of their experiments, which were of course, admittedly highly artificial, but they did get there, and occlusion of coronary arteries also produces an artificial condition. Now in the coronary arteries, when they are occluded, we have two sets of channels, coronary venous channels and the Thebesian vessels. It seems to me that either one of these might function as the source of the blood and the other for the return of the blood; it might be one, or it might be the other. Now if one considers this problem from the point of view of the dynamics of the heart, it has always been difficult for me to understand how blood would get in through the Thebesian vessels to nourish the heart. Dr. Wearn dismisses the work of Dr. Batson and Dr. Bellet as offering an unlikely explanation of this, but I should be very much interested to hear what pressure differences he believes develop in the ventricles which are superior to those which may arise from the auricle to get blood into the myocardium. Certainly, after systole begins one would think that the pressure within the myocardium itself would be at least equal to that of the intraventricular content, which would seem to me to be not a very favorable dynamic set-up for blood to enter through the thebesian vessels.

DR. WEARN, Cleveland, Ohio.—I have been asked a great many questions that I cannot answer. So far as I know, the answers to most of them are not known at the present time. Dr. Katz asked if there is a sudden change in the Thebesian vessels when they take over the function of the coronary arteries. The answer to his question would require knowledge of the size of the vessels before and after death.

I know of no way of getting such information. Even if one could look into the chambers of an intact heart the Thebesian vessels could not be seen. Their openings are usually buried beneath the trabeculae in the walls of the chambers. The fact is, if facts can be arrived at by deduction, that in the two cases cited, the thebesian vessels were the only channels by which the myocardium could get its blood supply under sufficient pressure to maintain it.

Now do the Thebesian vessels grow larger? Again I do not know. I do not know of any way of finding out, because you have not got them beforehand or afterward. They vary in size tremendously, from some with an opening of half a millimeter to those that are almost microscopic. You cannot see some of the openings and yet in injecting the hearts, one sees the dye ooze out through these particular places.

Some one else asked, "What blood pressure can the Thebesian circulation maintain?" I submit the two experiments to you, the heart of the woman with the luetic aortitis and that of the sailor, both of whom had complete closure of their coronary arteries and who had blood pressure enough to work and earn a living. The sailor could pull a rope on a boat and carry an armful of awning rods up a stepladder and put them up. That is sufficient, I should think, to show that the Thebesian circulation can maintain a normal blood pressure.

Dr. Wolferth's question brings up points on which opinions differ. Batson and Bellet's experiments have shown the presence of particles, injected into the femoral vein, in the cardiac veins. They believe that the particles got into the veins as a result of back flow from the right auricle, and from this evidence they conclude that back flow through the veins may furnish blood supply to the heart muscle.

I shall not attempt to explain how their particles got into the heart veins, but their conclusions are not acceptable to me. Their claims rest on a blood supply to the heart muscle, supported by pressure developed in the right auricle. The right auricle is capable of maintaining only a very low pressure.

The Thebesian vessels, on the other hand, open directly from the ventricular walls, and blood entering them would be under full ventricular systolic pressure. I have shown you actual histological sections which prove that the thebesian vessels open directly into the capillary bed. In other words they offer more direct channels to the capillaries than do the coronary arteries.

DR. KATZ, Chicago, Ill.—What was found at the autopsy in those cases of angina pectoris that died?

DR. LUTEN, Saint Louis, Mo.—May I ask Dr. White if he follows a more or less invariable rule as to the length of time he keeps a patient in bed after occlusion, even though all the evidence may indicate that the occlusion was in a small artery?

DR. ROBEY, Boston, Mass.—Is it not possible that some, perhaps many, of the more severe attacks of angina pectoris are due to thrombosis occurring in the finest branches of the coronary artery? The attacks are not severe enough to cause pericarditis, leucocytosis or even mild collapse. We know from hearts injected by Louis Gross and others that many of the old hearts show a great diminution in the finer branches of the coronary tree and they have probably become thrombosed. It is right to say that patients die of angina pectoris? Is not angina pectoris merely a symptom of some other condition?

DR. TALLEY, Philadelphia, Pa.—I have been interested in Dr. White's mention of respiratory difficulty without pain. A recent patient had no pain, but sudden great difficulty in respiration with leucocytosis, fever, characteristic electrocardiogram and later attacks of pulmonary edema. Even after two months on one floor, on two occasions one flight of stairs brought on attacks of acute pulmonary edema

that almost ended his existence. It was necessary to keep him on one floor for six months before he could do one flight with comfort. Since then he leads a comfortable but somewhat restricted existence.

DR. MORRISON, Boston, Mass.—I should like to ask if the degree of fall in blood pressure in acute coronary occlusion is of any prognostic value.

DR. MCGUIRE, Cincinnati, Ohio.—Have you any theory regarding the mechanism of the transient hyperglycemia which one finds following coronary occlusion? Have you observed a similar rise in the sugar content of the blood in cases of angina pectoris in which you believed that the coronary arteries were not occluded?

DR. WHITE, Boston, Mass.—Some of these are hard questions. As to the post-mortem examinations in our cases proved not to have coronary thrombosis, we have sometimes been astonished by the small amount of coronary disease or obstruction. Some of the patients, however, who died during an attack of angina pectoris had apparently good heart muscle with fair coronary arteries, and it is a mystery why they should have died.

Dr. Robey asked the question, "Do patients actually die of angina pectoris?" They die during an attack but we do not know what the mechanism of death is as yet; it is an interesting problem to be solved some day, but it is a considerable mystery. The death is not, apparently, like that of the Adams-Stokes syndrome with loss of consciousness and convulsions. Patients with angina pectoris die more quickly than that; they do not continue to live and have convulsions but they die at once. Why they die is a question that has not been answered; at least they die during attacks of angina pectoris.

As to whether smaller attacks of coronary thrombosis may occur to explain some of the severer attacks of angina pectoris, I do not think we can answer that. It is probable that minor thromboses can occur without reactions of fever and leucocytosis of any important degree, but generally it is easy to distinguish between the two, that is between angina pectoris and coronary thrombosis.

The length of time that I am accustomed to keep patients in bed after attacks of coronary thrombosis in the absence of any unfavorable signs or symptoms is a minimum of three weeks; if they have any unfavorable signs or symptoms, I require a longer rest in bed, sometimes months. We cannot be too careful and many people feel that three weeks are rather a short time. This depends on the individual case. There must be a long interval of convalescence; probably a few months of almost absolute rest at the beginning is a wise procedure if we want to establish a firm scar. I dare say that my minimum of three weeks may seem short, but some of my patients who are doing very well and who have survived for many years went through the relatively short period of absolute rest of two to three weeks followed by a convalescent period of some time after that before returning to moderate activity. Too long in bed is bad for the morale.

Diabetes was not common in my series, although it is occasionally found with angina pectoris; if we take a group of diabetic patients, we will find that angina pectoris is fairly common among them but diabetes is not correspondingly so common in groups of angina pectoris or of coronary thrombosis. I have little or no knowledge of the blood sugar content during coronary thrombosis.

The degree of fall in blood pressure is probably of some importance in cases of coronary thrombosis. It is of immediate importance, if extreme, in some patients who die very soon after the attack. A very low blood pressure lasting for weeks I think we may consider a bad prognostic sign, but many of my patients who survived for years had a considerable drop in pressure at first with a return to normal after a few hours or days.

Book Review

HERZ - UND KREISLAUFINSUFFIZIENZ. EIN KURZES SYSTEM DER STÖRUNGEN IM KREISLAUFAPPARAT. By Dr. K. F. Wenckebach. EM. Vorstand der 1. Medizinischen Universitätsklinik, Wien. 120 Seiten. Mit 7 Abbildungen. Dresden und Leipzig. Verlag von Theodor Steinkopf, 1931.

In view of the ever increasing mass of medical literature, it is quite proper to inquire whether a particular new publication is justified and will fulfill a need. If one applies this test to the admirable monograph by Wenckebach, one may answer the question unequivocally in the affirmative. It is necessary for teachers of medicine to present the results of laboratory investigations and their clinical scientific observations in such a form that he who runs (i.e., the practitioner) *will* read, understand and apply the new knowledge in his practice. Wenckebach has had this objective constantly before him and has reviewed the recent advances made in the clinical science of the circulation in an admirably simple way. It is noteworthy that he has been willing to follow the trend of the times in cardiovascular disorders and turn his attention from arrhythmias which were the main subject of the brilliant investigations of his earlier career to the hydrodynamics and mechanics of the circulation as a subject of contemporary interest with which the general practitioner has not as yet familiarized himself. Moreover, his viewpoint has been broad, his critical judgment excellent, and in his references to the literature he has done justice to the English, American, Dutch and Belgian workers in this field.

The monograph opens with an important introduction on the effects of local obstruction or interference with the blood stream, and a consideration of the reasons for the violent opposition of James Mackenzie to the "back-pressure" theory. Following this, the dynamic laws of the heart are discussed, and here a more elaborate exposition, even at the expense of more space, along the lines of Starling's Linacre lecture on the "Law of the Heart" would have helped to elucidate this rather difficult subject.

The subject matter of the monograph proper is divided into two main parts, both dealing with the laws of cardiac and circulatory insufficiency. In the first part are described the hydro-mechanic and dynamic consequences of various obstructions to the circulation, "Ceteris Paribus," i.e., other conditions, however, remaining the same; whereas in the second part entitled "Ceteris non Paribus," those factors are discussed which may alter or entirely change the events in the mechanism of the

circulation described in the first part—irrespective of whether such factors are helpful or detrimental to the circulation.

In the first part Wenckebach discusses disturbances of circulation of cardiac origin, including left heart failure, stasis in the pulmonary circulation, disturbance of the right side of the heart such as right heart failure, reciprocal relation of pulmonary congestion and hepatic congestion, reciprocal action of hepatic and renal congestion, tricuspid insufficiency, auricular fibrillation, the type of stasis due to pericardial disease, and circulatory disturbance resulting from equal damage to both sides of the heart. Then under the general topic of obstruction in the greater circulation, Wenckebach takes up the involvement of the large veins, and of the smaller and smallest veins, the phenomena of collapse and of shock, the capillaries, the arterial system including blood pressure, hypotension, hypertension, Hochdruckstauung (congestive hypertension) of Sahli, Stauungshochdruck, Heberden's angina pectoris, coronary circulation, and the reciprocal action of aortic and pulmonary congestion.

The second part of the monograph, with its caption, "Ceteris non Paribus," is devoted to a discussion of other factors which may modify the dynamics of the circulation previously described. The following are among the subjects treated: mechanical aids to the circulation, water exchange, metabolism, edema, metabolism and circulatory insufficiency, minute volume, stroke volume, oxygen debt, blood volume, short circuits in the circulation, velocity of circulation and cyanosis, cardiac rate and rhythm, digitalis therapy, dyspnea and its relation to the respiratory centre.

In one word, the book is a review of the contemporary views on the circulation and circulatory failure, written for advanced students and practitioners by an investigator and teacher of medicine who has himself taken an active part in the astonishing progress made in the study of the heart from the time when the chief interest was concentrated in the arrhythmias to the present period when the dynamics of the circulation as a whole appears to hold the centre of the stage. In this work Wenckebach has tried to bridge the great gap between the clinician and the experimenter, and believes the two may meet and by cooperation may make still further contributions to science, to the benefit of the practice of medicine.

B. S. O.